VOLUME 13
NUMBER 1
JANUARY, 1960

ORAL SURGERY
ORAL MEDICINE
and ORAL PATHOLOGY

EDITORIAL

COMPOSING A SCIENTIFIC ARTICLE*

STUDENTS, particularly graduate students, are urged to read, but they seldom are taught to write. A time may arrive, though, when interesting and instructive material is available. A series of cases of an uncommon disease (at least three are needed) may stimulate you to write a paper. Perhaps you can present the results of original experiments, the conclusions of a research project, or the observations of unusual and different disease symptoms or signs which have escaped publication. Do not write a paper, however, unless you feel that its publication will be a significant addition to the literature and will benefit your professional colleagues and, indirectly, your fellow men. It is well, in addition, to review the literature on the contemplated subject before writing the article to determine whether or not your project will enhance or negate the findings of others.

You may have an urge to sit down and write the article in one evening, have it typed the next morning, and rush it to the editor of an appropriate journal. Editors get far too many poorly written papers, and if you think it is their job to rewrite them you are mistaken. Do not expect an editor to do ghost writing. His task is to select good papers of interest to the readers of his journal, make minor changes in the text, and see to it that the accepted terminology is used.

The rewriting should be done by the author of the article. "Eine Arbeit muss verarbeited werden" said one of my Swiss professors. A period of gestation is required. This, in the case of elephants, is two years; the human brain may do it more quickly. Before you begin to write, you should have a clear concept of what you want to say to the reader of your article.

First, clear the thoughts in your mind, ponder on the presentation of your material and how it should be shaped, and after careful consideration seize your pen and write. You may now write fully about the principal subject, and in a few hours have the *pièce de résistance,* the meat of your article, before you.

*Inspired by "How to Write an Article" by Peter Quince, *Perspectives in Biology and Medicine,* winter, 1959.

This is, however, only a draft, a beginning which may not be usable in its present form, but you have benefited from exercising your mind. When rereading the thoughts which you have put down so far, you may find that your subject has opened in many directions. You may have strayed into unprofitable side lines. You may find that the article has to be completely revised; perhaps it should be scrapped and you should begin anew.

Put the paper aside for a few days and then read it again. Unnecessary discussions may be deleted, or you may want to add important new thoughts. Perhaps you will get stuck. Don't worry; time will be your ally. Sleep on it. Your subconscious may come to your rescue; forces stronger than you know may come to your aid. Thus, you may gather a lot more fragments which may be fitted in different juxtapositions so that you may have to consider entire paragraphs again and again. Have you fitted the parts together as you would a jigsaw puzzle? Are there gaps which need to be filled in? Is the picture clear, concise, well demarcated? Or are there fuzzy areas? You may find a certain amount of redundancy in your manuscript, and redundant statements should be eliminated. Hold on to the phrases which best express your idea—the ones that have punch.

Now we come to the reducing of the material. You may find examples of esoteric neologism, as you may have become involved in chronic logorrhea instead of presenting your material in simple language. Cut out a few dependent clauses; alter your punctuation, if necessary. Can't those three adjectives be replaced by one? Are there ambiguous statements in your text? Are your statements too all-inclusive, too positive? Never say "cannot," and be careful if you say "always." Check the dictionary for the exact meanings of words and be sure to watch for common errors found in medical articles. Many authors use the words "the case" when they should say "the patient." Saying that the patient has a temperature means nothing; he may have a fever or a raised temperature.

A sigh of relief may be permitted at this time; you have worked hard. Take a rest and then read your text once again; even now, glaring imperfections which you would not have noticed a week ago seem to occur in many paragraphs and may mean additional work. Have you made a skeleton of your paper to see if it is properly organized and in logical sequence?

If reorganization is necessary to produce order out of chaos, it may be done with scissors and paste. Whole paragraphs can thus be relocated or a substitution glued into place. This, however, may take just as much time as a wearisome transcription.

The time has finally arrived to write the introduction and the conclusion to your article. The introduction should state why the article should be published, what new findings it contains, and how your findings differ from others already published. Do not fill pages with accepted facts and historical developments. Remember, you are not writing for students; you are addressing yourself to men of experience. There should be added a bibliography which lists

authors who have published similar material, and proper credit should be given, by means of accurate and enumerated references, at the end of the article in the manner customarily prescribed.

In most instances, a summary should be added to give those who are engaged in related fields a chance to see whether the article is of interest to them. In this should be stated the problems, the methods used to arrive at a conclusion, the results obtained, and the deductions made. Such a summary will also serve as a résumé that can be used as an abstract by journals reviewing the current literature or translated into other languages.

Finally comes the time to try your article on the dog, or perhaps your wife is good natured and will allow you to read it to her. Be careful. If she majored in English, she may tear it apart again. Wives are out unless they can temper their criticism with praise—say, in the proportion of one to four. A senior author who may have his name attached to the enterprise should certainly be made responsible for the quality of the manuscript, but if you have no senior author as a collaborator, an expert in the field may be asked to review your masterpiece. Your reader may spot ambiguities, awkward sentence construction, and grammatical howlers. Take the criticism, make the final changes, retain your humility and good nature, and boy you have done well!

Thus, suddenly your paper is perfect and there is nothing more to be done, except to find a journal which will accept it. Remember, however, that the last word is spoken by the editor.

K. H. T.

OPERATIVE ORAL SURGERY

. . . . . .

PROGRESSIVE ATROPHY OF THE MANDIBLE COMPLICATED BY FRACTURES: ITS RECONSTRUCTION

Kurt H. Thoma, D.M.D., Dr. med. dent. h.c., F.D.S.R.C.S. (Eng.), Hon. F.D.S.R.C.S. (Edin.), F.A.C.D., Brookline (Boston), Mass.*

PROGRESSIVE atrophy occurs after extraction of the teeth and affects first the alveolar process and, in some cases, the basal bone of the mandible. It occurs mostly when the teeth have been lost because of advanced periodontal disease. Cases of localized atrophy occur if excessive pressure is exerted by a partial denture.[1] The patients affected by severe general atrophy of the jaw usually present great problems in denture construction, but today their masticatory function may be satisfactorily restored by means of implant dentures. When the resorptive process has progressed to the state where only a very thin span of bone remains, however, there is danger of pathologic fracture. Fracture may occur if the bone is further weakened by the use of screws to attach an implant, as in the case to be reported. In addition, one must consider that the bone becomes more brittle with age and therefore measures must be taken to reinforce the remaining mandible. Otherwise, complete atrophy may result with the entire bone disappearing, as in a case reported by me several years ago.[2]

The following case illustrates an unusual condition in which spontaneous fractures of a very atrophic mandible were treated and further trouble prevented by means of bone grafts, after which the patient's teeth were restored by means of a new implant denture.

CASE REPORT

R. S., a 47-year-old housewife, was referred to me because of spontaneous fractures of the mandible with gross displacement. She had the appearance of a patient with micrognathia due to retrusion of the chin. She stated that since the loss of her teeth nineteen years earlier, the ridge of her mandible had undergone resorption to the extent that she was unable to wear her denture. An implant denture was recommended, and this was inserted in New York. The implant, however, had to be removed three weeks later

*Professor of Oral Surgery, Boston University; Chief of Oral Surgery, Brooks Hospital, Brookline (Boston), Massachusetts.

but was replaced by another one. Six months later a swelling appeared on the left side; this was painless but was associated with a discharge of pus where the posterior post projected through the gingiva. Examination had revealed abnormal motility, and a roentgenogram disclosed the presence of a fracture. Treatment with antibiotics caused improvement, but five months later a swelling developed on the right side. There was no suppuration, but the roentgenogram showed that here also a fracture had occurred. The implant was then removed and antibiotic therapy was resumed. This caused a gradual improvement. The patient was told that a pseudoarthrosis had developed at the fracture site.

Examination.—When the patient was referred to me, she brought with her roentgenograms, which showed three fractures of the mandible, presumably at the site where the screws had been inserted for the attachment of the implant. There was displacement downward of the symphysis. Additional films showed atrophy of both maxilla and mandible, with three fractures in the latter. There seemed to be partial consolidation of the fracture on the left but no union on the right. The horizontal ramus seemed to be displaced by the muscles attached to it (Fig. 1).

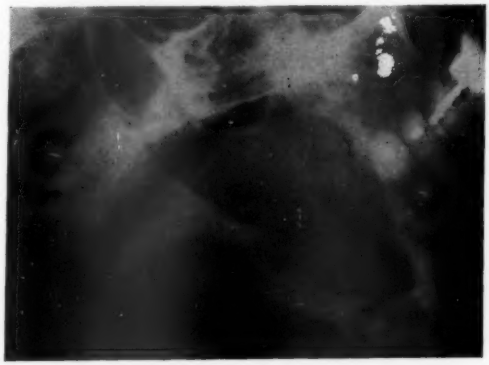


Fig. 1.—Progressive atrophy of mandible with three spontaneous fractures.

The patient's general health was satisfactory. Roentgenograms were taken of the skeleton. The blood chemistry showed serum calcium 10.7 mg. per cent, phosphorus 3.0 mg. per cent, and alkaline phosphatase 5.5 units. The physical examination yielded no important information except for rheumatoid arthritis for which the patient had received synthetic steroid cortisone preparations. She stated that she was allergic to penicillin and Demerol.

Surgical treatment of the fractures with rib graft reconstruction of the mandible was recommended. The patient contracted influenza, for which reason the operation had to be delayed for five weeks. During this time she was placed on a high-protein, vitamin-reinforced diet with the protein and vitamin supplementation in the form of Meritene to be taken two or three times a day.

First Operation.—The operative procedure was performed following induction of intravenous anesthesia with endotracheal administration of nitrous oxide and oxygen. The operation consisted of two parts—(1) the excision of the left and right eighth ribs by Dr. M. Osborne and (2) the jaw operation. The latter was first carried out on the right side.

After careful preparation of the skin, an incision was made extending from the midline beneath the chin to behind the angle of the jaw. The displaced fracture was

exposed and the fragments were separated (Fig. 2). It was interesting to note that the progressive resorption of the mandible had proceeded to such an extent that the mandibular canal had become eliminated, the neurovascular bundle being outside the bone as

Fig. 2.

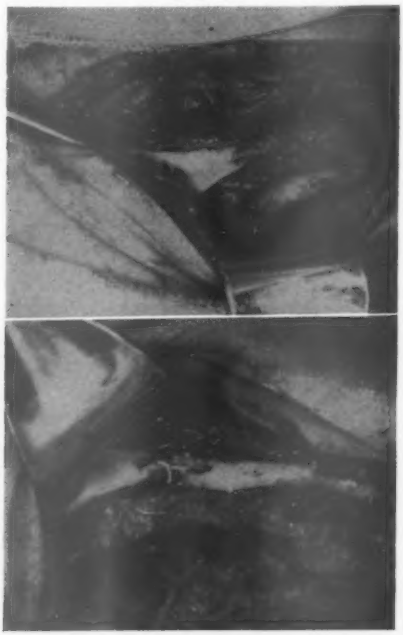


Fig. 3.

Fig. 2.—Exposure of fracture on right side with neurovascular bundle lingual to mandible.
Fig. 3.—Reduction and wiring fixation of fracture.

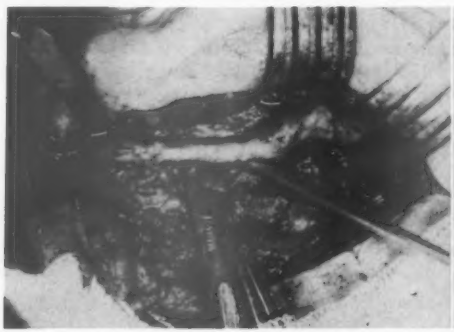


Fig. 4. Fig. 5.

Fig. 4.—Left side of mandible with malunion of fracture.
Fig. 5.—Fraise.

shown in Fig. 2. After the ends were freshened, they were properly positioned and fixed by transosseous wiring (Fig. 3). In order to accomplish this, the mandible was pulled

forward by means of a nerve hook inserted into the symphysis. The external surface of the mandible was freshened with an engine-driven fraise (Fig. 5) and a drill, after which a piece of the left rib, split lengthwise, was cut to the right length. The fraise was again used to make a groove at the inner side of the rib to receive the atrophic mandible. To curve the rib in the canine region, three V-shaped vertical cuts were made on the inner surface; this allowed bending without actually producing a greenstick fracture. Since the ribs were thinner than anticipated, a second piece was made ready to apply to the lingual side of the mandible. After the ribs were properly placed, several holes were drilled through the ribs and the mandible and stainless steel wires were inserted. The wires were twisted to hold the ribs and mandible together, after which the posterior part of the wound was closed.

Fig. 6.

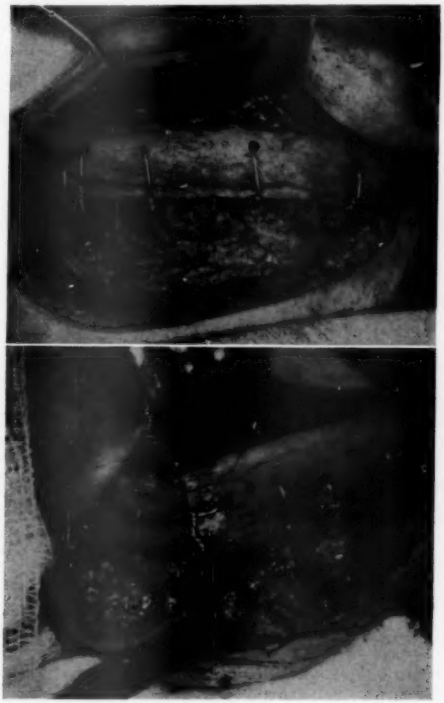


Fig. 7.

Fig. 6.—Split rib attached to outside and another inside the mandible with transcircumferential wires holding the bones together.
Fig. 7.—Left side and symphysis.

The other side of the mandible was then exposed by continuing the incision from the midline to the left angle of the jaw. The same procedure was carried out here as on the right side, except that the partly healed fracture was not revised (Fig. 4). Two sections of the right split rib were then fitted to each side of the thin span of bone and wired into position, building out the left angle which was depressed medially by extending the graft posteriorly. The anterior ends then were trimmed and wired together. Since they formed a Gothic arch, two transcircumferential wires were added to each side of the midline to flatten out the symphysis, as shown in Fig. 8. Fig. 6 shows the reconstruction on the right side, and Fig. 7 shows the reconstruction on the left side and symphysis.

The patient did well postoperatively except that she expelled all subcutaneous catgut sutures, since she seemed to be allergic to catgut. The Dermalon sutures were removed on the sixth and eighth postoperative days. There was little postoperative swelling; the patient had some ecchymosis of the neck and very little discomfort. She was kept on Tetracyn, 250 mg. every four hours, and the high-protein, vitamin-reinforced diet (Meritene) was continued. After four weeks she was discharged to go home. She visited the Brussels International Exhibition and ten months later presented herself for re-examination. She was perfectly satisfied with the result of the operation, but she felt that her chin was not prominent enough. I admitted her to the hospital for a genioplasty.

Second Operation.—The genioplasty consisted of the resection of a segment of bone from the iliac crest by Dr. M. Osborne and the attachment of this bone to the jaw.

Following induction of intravenous Surital sodium anesthesia with nitrous oxide and oxygen administered through an endotracheal tube, and after the usual skin preparation, Dr. Osborne resected a piece of iliac crest.

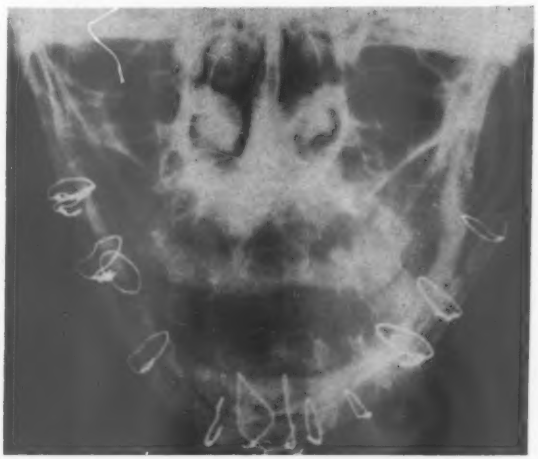


Fig. 8.—Postoperative posteroanterior view.

I placed new drapes for the operation on the jaw. The old scar in the anterior part was excised in order to expose the rib graft previously inserted. The bone here appeared in very good condition. The rib grafts and mandible had been completely consolidated. The wires that had been inserted were covered with scar tissue which had to be scraped away to expose the metal. Five wires were cut and removed. Following this, the iliac bone was shaped with rongeurs and filed so that it could be butted against the mandible. An oblique hole was drilled on each side of the symphysis to correspond with two holes drilled in the bone graft. Stainless steel wire was drawn through these holes and used to attach the iliac crest bone to the lower border of the rib grafts (Fig. 9). The result seemed to be satisfactory. The crest of the graft formed the periphery of the new chin (Fig. 10). The skin, therefore, was drawn over it, after which the wound was closed with linen subcutaneous sutures and interrupted vertical mattress Dermalon sutures. The patient did well postoperatively as far as the genioplasty was concerned. The sutures were removed on the fifth, sixth, and seventh postoperative days. She was then discharged, to be followed by Dr. Osborne until she had completely recovered from the hip operation.

The patient was seen several times for postoperative checkups. Six months after the last procedures, the rib grafts looked well on x-ray examination, though the graft to

Fig. 9. Fig. 10.

Fig. 9.—Genioplasty. Iliac crest attached with two wires.
Fig. 10.—Postoperative roentgenogram.

Fig. 11.

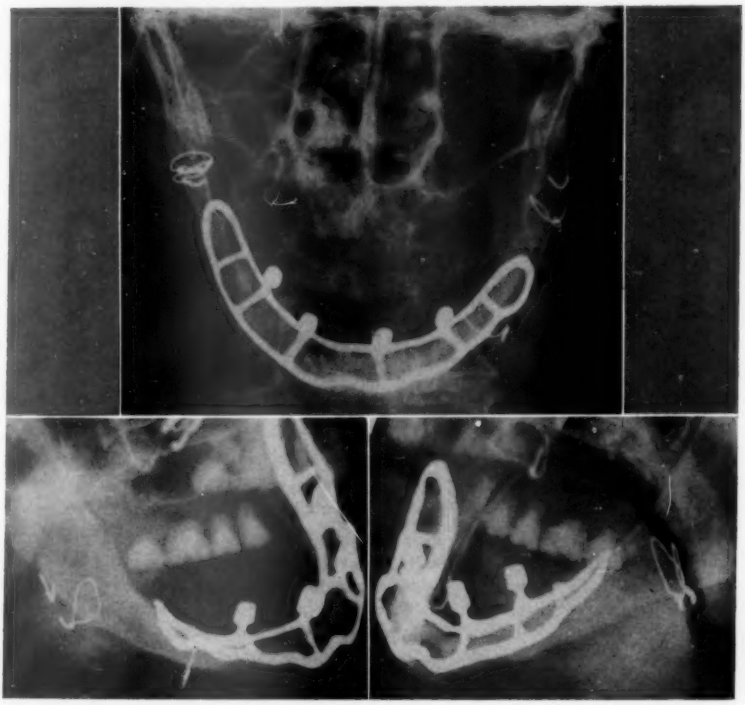


Fig. 12. Fig. 13.

Figs. 11 to 13.—Implant denture in place. Most of the wires have been removed.

the chin showed some evidence of resorption. The patient was pleased with the reconstruction of her jaw, but she still was unable to masticate solid food. She brought up the question of having a new implant denture made. I referred her to Dr. J. Barron for the preparation of the prosthetic replacements, after which she was readmitted to the hospital for the first stage of the implant procedure—the bone impression.

Third Operation.—For protection, the patient received a broad-spectrum antibiotic. The operation was performed again under intravenous Surital anesthesia with gas and oxygen administered through an endotracheal tube. After the usual thorough preparation of the face and oral cavity, an incision was made on the crest of the mandible extending from the ascending ramus on one side to that on the other. A cross incision was made at each end, with a labial extension in the midline. The detachment of the mucoperiosteum presented some difficulty since it seemed firmly attached to the ribs, and it was necessary to use sharp dissection in many places in order to lay the bone bare. Some of the wires which had been used to hold the ribs and mandible together were removed, the bones having consolidated completely. After the bone impression was taken, the wound was irrigated with warm saline solution, sterilized with tincture of Zephiran, and closed with a continuous locked silk suture. A pressure dressing was applied between the jaws. The patient was discharged from the hospital on the third postoperative day.

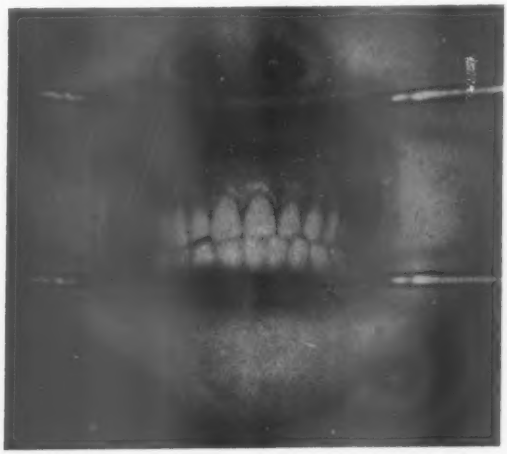


Fig. 14.—Upper denture and lower implant dentures constructed by Dr. Joseph Barron of Boston.

The patient did well after the first stage of the operation. The sutures were removed on the sixth postoperative day and she was readmitted six weeks later for the second stage of the procedure, the implant having been made ready in the meantime.

Fourth Operation.—The second stage of the procedure, the insertion of the implant, was again performed under intravenous anesthesia. After the mouth was thoroughly prepared with tincture of Zephiran, a new incision was made from one retromolar area to the other without accessory extensions. The bone was laid bare, and the sterilized implant was pressed into place. It appeared to fit well and to resist removal. The mucoperiosteum was closed over the implant with interrupted silk sutures, and around the four posts with horizontal mattress sutures so as to adapt the gingiva close to the protruding metal.

The patient made a good recovery, with little or no swelling or discomfort. After the sutures were removed, she was discharged and instructed to return in one month for a

postoperative checkup. She was also to see Dr. Barron for the construction of her dentures. Postoperative roentgenograms are shown in Figs. 11, 12, and 13; Fig. 14 shows the new upper denture articulated with the implant denture.

SUMMARY

A case of severe atrophy of the mandible with three spontaneous fractures has been presented. The treatment of the fractures and the reinforcement and reconstruction of the mandible with bone grafts have been discussed. I have also described the restoration of masticatory function in this patient by means of a new implant denture.

REFERENCES

1. Thoma, K. H.: Atrophy of the Mandible, ORAL SURG., ORAL MED. & ORAL PATH. 4: 1477, 1951.
2. Thoma, K. H.: A Case of Progressive Atrophy of the Jaws, J. Bone & Joint Surg. 15: 494, 1933.

1146 BEACON ST.

CHRONIC UNILATERAL DISLOCATION OF THE MANDIBULAR JOINT TREATED SURGICALLY BY A HIGH CONDYLECTOMY

Report of a Case

Robert W. Christensen, D.D.S., Pasadena, Calif.

THE treatment of intermittent or constant dislocation of the mandibular joint may be required only rarely, but it can offer problems to the oral surgeon whose professional services are sought. Single episodes of acute dislocation of this joint occur with some frequency, but the treatment of this condition is well known. Treatment by manual reduction of the dislocation will usually suffice, and a repeat attack, if it occurs, will respond well to this treatment.

The patient in whom dislocation of the mandibular joint becomes so frequent that periods of rest between occurrences are very short, if they exist at all, may present a problem to the practitioners diagnosing and treating the case.

In the case to be presented, there was a long history of recurrence of the dislocation of one mandibular joint to the point where continual dislocation was brought about, not by the patient's physically and voluntarily opening the jaw but through spasm of the external pterygoid muscle. Such a case may present a problem in diagnosis and treatment, but when the problem has reached this degree of severity, the patient is in utter agony from the pain.

Subluxation or dislocation of the mandibular joint may be due to a strain or weakness of the capsule or ligaments caused by an anatomic variance of the joint, an acute injury, malocclusion or improper jaw function, or an individual episode of hyperactivity of the mandibular joint during anesthesia or yawning.

If dislocation becomes habitual, we might find that a reflex nerve pattern involving the sensory nerve fibers of the capsule or the posterior attachment fibers of the meniscus and the motor nerve fibers of the muscles of mastication might be placed in a state of irritability. If this reflex arc of neural irritability should include the motor nerve fibers of one external pterygoid muscle, we might expect to see a downward, forward pull of the involved condyle from its position in the glenoid fossa. In the case to be presented, there was an involuntary opening of the molar teeth on the involved side, even when the anterior teeth were held, by traction, in centric occlusion.

If we consider the anatomy of the joint, with particular reference to direction of muscle pull, we will find that the powerful lower head of the external pterygoid muscle is the only muscle involved directly with the mandibular

joint which can, by itself, pull the condyle downward and forward. The large lower head of the external pterygoid muscle attaches to the capsule and especially to the anterior surface of the condylar head and is in total agreement with the direction of mandibular movement (Fig. 1). The pull of the weaker upper head of the external pterygoid muscle, with its attachment to capsule and articular disc, however, is directed upward, forward, and inward. Besides the possible strain of the capsule or ligaments, the pressure of the condyle causing injury to the vascular and nerve-innervated loose connective tissue fibers which attach the posterior surface of the disc to the capsule may be the initiator of the reflex nerve arc which places the external pterygoid muscle in contracture to relieve the pressure of the condyle.

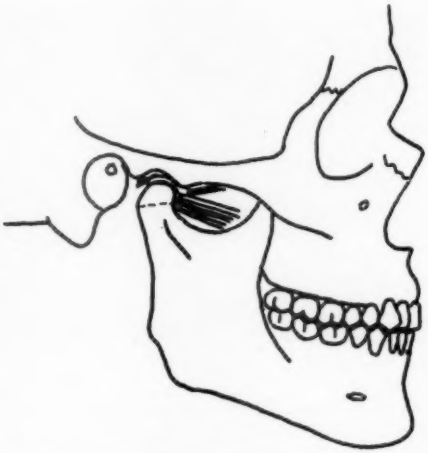


Fig. 1.—Diagram showing the attachment of the larger external pterygoid muscle to the head of the condyle and the weaker section of the external pterygoid muscle to the articular disc.

The treatment in such a case should be directed toward reducing the reflex irritability. This may be accomplished by one or more treatment methods, depending on the individual case. Treatment may consist of (1) equilibration of occlusion or correction of malocclusion, (2) bite-raising with occlusal splints, (3) limitation of function, (4) use of drugs, such as analgesics, muscle relaxants, sedatives, anti-inflammatory agents, or sclerosing solutions, (5) x-ray therapy, or (6) surgery. The treatment of any given case will have to be selected for the particular patient involved.

In the case to be presented, many of these forms of therapy were instituted until finally surgery was indicated and was successful in correcting or controlling the problem.

CASE REPORT

On May 21, 1957, I was called by the resident physician in the emergency section of Huntington Memorial Hospital to examine and treat a patient with an acute dislocation of the mandibular joint.

Present History.—The patient was a 32-year-old white woman who was on the hospital staff as a registered nurse. She was on duty as a floor nurse when, during a period of yawning, her jaw dislocated. She had been brought to the emergency room and was immediately given 100 mg. of Demerol intramuscularly by the resident.

Examination.—In spite of the sedative, the patient was in acute distress because of a unilateral complete dislocation of the left mandibular joint. The patient was of large stature physically and in generally good health. She believed that she was in her second month of pregnancy, but she had not sought obstetrical consultation.

Her mandible was open approximately 3.0 cm. in the incisor area and was shifted 1.5 cm. to the right side (Fig. 2). Nothing remarkable about her dental condition was noted at this time. By palpation over the left condylar region, the lateral protuberance of the condylar head was felt extruding laterally. The condyle was lying anterior to the articular eminence out of the glenoid fossa.

Fig. 2.—Patient with a unilateral dislocation of the left condyle of the mandible.

It was impossible for the patient to move or close her jaws voluntarily. Palpation disclosed that the right condyle was lying in its anatomic position in the glenoid fossa. No pain or pressure was noted over the right mandibular joint, but pain was very marked over the left condylar area.

Emergency Treatment.—After the patient was questioned and any probability of known drug allergies was ruled out, the preauricular area over the left mandibular joint was cleansed with soap and alcohol and draped. A skin wheal was raised with Xylocaine 2 per cent in a 30 gauge needle. When this had produced the desired anesthesia, a periarticular injection of Xylocaine was made with a 27 gauge needle. When the area was anesthetized, a manual reduction of the dislocation was accomplished in a manner that I have found very useful.

The index finger of the operator's right hand is placed in the patient's mouth and is directed laterally to the left maxillary first molar along the gingival margin. The finger is pressed posteriorly until the anterior margin of the coronoid process is palpated. The coronoid process of the mandible will be anterior and closer to the lateral surface of the tuberosity; when the index finger is forced between the coronoid process and the maxilla, the coronoid process will be forced laterally and posteriorly, causing the condylar head to re-enter the glenoid fossa. It may be necessary to use the tip of the finger as a fulcrum against the posterior surface of the tuberosity and to bend the proximal portion of the finger laterally to accomplish this.

In this instance, the dislocation was reduced and then an intra-articular injection of 25 mg. Hydrocortone acetate was given to aid in reducing posttreatment edema, inflammation, and pain in the joint. The patient was given a prescription for codeine to be used for pain and was asked to return to my office for evaluation and observation on the fourth day.

The patient was unable to keep her office appointment but was rescheduled for two weeks later. One week after the original treatment, I was again called to the hospital when the patient suffered another identical dislocation of the left mandibular joint. I treated this dislocation in the same manner as the earlier one.

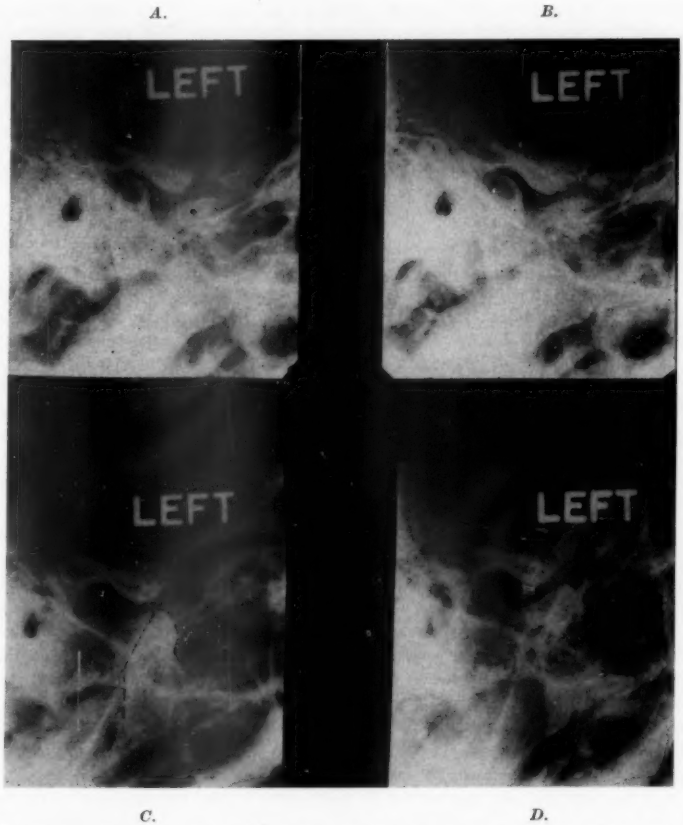


Fig. 3.—Roentgenograms showing patient's condyle in closed (*A*), relaxed (*B*), open (*C*), and dislocated (*D*) positions.

On June 4, 1957, the patient was examined in my office. At this time her jaw function appeared normal except that she voluntarily restricted the vertical opening of the mandible. This was a self-induced restriction of function to approximately 2.5 cm. opening, since beyond that point the patient would experience a dislocation. She had a severe overbite in the maxillary incisor area. Her teeth were in good repair with good restorative dentistry; there were some areas of marginal caries. There was slight clicking in the left joint, and there was some tenderness to palpation over the joint. Otherwise, the oral examination was not remarkable. The patient was referred to the oral roentgenologist for full-mouth periapical dental films and open- and closed-temporomandibular-joint films. During the x-ray examination the patient's jaw dislocated, and roentgenograms were taken of the joint at this moment (Fig. 3). Following the x-ray series the dislocation was reduced.

Over the next three-week period, the patient's left condyle dislocated twice again. Once she was treated by a physician at a hospital in another community, where a great deal of difficulty was experienced in reducing the dislocation.

On July 1, 1957, at 1 A.M. the patient was brought to my office by her husband. Again her jaw had dislocated. The treatment was similar to the previous times, except that the patient's pain was more severe and uncontrollable. I gave her 100 mg. Demerol and injected Xylocaine into the periarticular tissues. When the dislocation had been reduced and the patient's husband and I were discussing the problem, we saw her facial expressions become tortured and noticed a jerking motion of the mandible as it was involuntarily becoming dislocated. I quickly reached over to apply pressure to the chin, but to no avail. There was occurring an unquestionable contracture or spasm which was exerting massive force. As I attempted to reduce the jaws manually, I could see the posterior occlusion on the left side open approximately 1.0 cm. with the anterior teeth still close together. As quickly as possible, more Xylocaine was injected around the joint and also into the pterygoid muscles through the sigmoid notch and from an intraoral approach. Nothing seemed to help the patient so far as pain or myospasm was concerned. Attempts were made to place wires around the molars and premolars and to apply wire immobilization, but it became apparent that the teeth would be extracted or the wires would break before all could be twisted.

The decision was now made to admit the patient to the hospital. There, after induction of general anesthesia with intravenous Pentothal sodium–curare with nasoendotracheal gas and oxygen, the patient's jaw was successfully immobilized with intermaxillary steel wires.

Following the patient's recovery from anesthesia, the spasms continued at intervals of every twenty to forty minutes and lasted from thirty to sixty seconds each. The pain was severe. Morphine sulfate, ¼ gr., was given intramuscularly every two to three hours. Nembutal, 1½ gr., was given every four to five hours for sedation. The patient was given intravenous feedings of Trinidex with 5 per cent dextrose in one liter of normal saline with 500 mg. Berocca C added daily. Liquids were forced orally. A muscle relaxant (Flexin, 500 mg.), dissolved in water, was given orally every four hours. No relief was obtained.

Past History.—There was nothing remarkable or contributory in the patient's past general history. Her jaw problem started two years previously when she was on active duty with the United States Naval Nurse Corps at Corona Naval Hospital. During a routine dental examination in April, 1955, the patient experienced her first dislocation of the left side of her mandible. The oral surgeon in the hospital reduced the dislocation, which occurred when the patient attempted to open her jaws farther than was comfortable for her. This was the first known incident of joint disturbance. For the following few days the patient experienced some tenderness in this area during normal functions of her jaws.

One month later the patient was again in the dental clinic of the Naval Hospital, having necessary dental restorations, when her left condyle again dislocated. The dislocation was treated, and the postoperative discomfort lasted approximately ten days.

Two months later the patient yawned, and again the same unilateral dislocation occurred. This was treated by the oral surgeon, and the patient was given an appointment to return to the dental clinic one week later for evaluation of her joint problem. At that appointment full-mouth periapical films and right and left mandibular joint films were taken. It was decided that, since the patient had a marked malocclusion and overbite, equilibration of occlusion with reduction of cuspal interference was indicated. This was done in several sessions. Study models of the dentition were made and used in accomplishing this.

The patient continued to have episodes of dislocation of the left condyle at roughly monthly intervals. Occlusal splints were constructed by the prosthodontist to raise the posterior occlusion. These splints were worn by the patient for three months with no relief from dislocation and with more pain in the left mandibular joint.

In June, 1956, during the mastication of a normal meal, a severe and sharp pain was felt in the left condylar area and dislocation of this joint followed. The patient was admitted to the Naval Hospital and, under general anesthesia, the jaws were immobilized by intermaxillary elastic traction after reduction of the dislocation. This was continued for four weeks, with the patient taking only liquid nourishment. Hypodermic injections of codeine

were given to relieve pain. Following this interval, the elastic traction was removed and the patient was cautioned to take only soft foods and to limit the excursions and opening of the mandible.

In October, 1956, the dislocation repeated itself and the patient was transfered to the United States Naval Hospital in San Diego for evaluation of her mandibular joint problem and for possible further treatment or medical discharge. Again her jaws were immobilized for three weeks. In November of the same year she suffered another dislocation and at this time the treatment involved the intra-articular injection of sodium morrhuate in an attempt to reduce any hypermobility of the joint. This was repeated in January, 1957. During the next four months the dislocation occurred twice and was subsequently reduced. In May, 1957, the patient was given a medical discharge from active duty because of her unusual joint problem. She came to Pasadena to engage in private duty nursing in one of the local hospitals.

General Physical Findings.—The general physical examination revealed no remarkable findings or contributory factors. A neurological consultation was secured, and Dr. Robert Pudenz, the neurosurgeon, found no evidence of definite neurological pathology. The patient was 5 feet 7 inches tall and weighed 137 pounds. Her blood pressure was 128/70, her pulse rate was 76, and her respiration rate was 20. Her general physical condition was normal. The patient stated that she was in her second month of pregnancy and had periods of hyperemesis gravidarum.

Laboratory Findings.—A hematologic examination on July 1, 1957, showed 4,350,000 erythrocytes and 7,300 leukocytes, of which 59 per cent were polymorphonuclear neutrophils, 34 per cent were lymphocytes, 4 per cent were eosinophils, and 3 per cent were monocytes. Bleeding and clotting times were normal. Blood calcium and phosphorous levels were normal. Urinalysis was normal.

Diagnosis.—The diagnosis was acute spasm of the left external pterygoid muscle with chronic left mandibular joint derangement. The course until July 4, 1957, was one of torture for the patient, despite the administration of opiates, sedatives, and muscle relaxants.

On July 4, 1957, it was decided that the head of the left mandibular condyle should be removed surgically. It was hoped that, with the removal of much of the area of attachment of the fibers of the powerful head of the external pterygoid muscle, the downward and forward pull of the left mandibular condyle would be minimized and perhaps prevent the dislocation. Also, with the loss of tension or pressure on the capsule and disc, I hoped that the self-perpetuating neurological reflex pattern might be broken and thus prevent spasm in this muscle and allow more normal masticatory function.

Operation.—On July 5, 1957, the patient was given routine premedication, prepared for condylectomy, and taken to surgery. After intravenous Pentothal sodium anesthesia was started, the intermaxil'ary traction was removed and the nasoendotracheal intubation was completed. The patient was given Pentothal sodium–Anectine intravenously with naso-endotracheal gas-oxygen anesthesia. The left side of the face, neck, and head was prepared and draped in the usual manner. Sterile cotton was placed in the ear. A slightly angulated preauricular incision was made with a scalpel through the skin and superficial fascia. The incision was contoured to just enter the ear and obscure any postoperative scar. Its length was approximately 4.0 cm., and it extended from the upper to the lower attachment of the ear. The skin flap was elevated by sharp dissection in an anterior direction, and the flap was sutured forward to the skin of the face. By sharp dissection, the incision was carried deeper along the anterior surface of the cartilage of the ear to the level of the zygoma. By following very closely to the cartilage of the ear, the branches of the facial nerve were avoided, the dissection was fast, and bleeding was minimal. The few bleeding points that were dissected were grasped with hemostats and electrocoagulated. At this point the fibers of the masseter muscle are incised from the zygoma and the capsule, and now the posterior border of the masseter muscle and the parotid were retracted, exposing all of the capsule of the joint (Fig. 4). An inverted "L" incision with its angle placed in the superior

posterior section of the capsule was now made (Fig. 5). The horizontal portion of the incision was just below the articular disc. The vertical incision was extended to the neck of the condyle, and now the apex of the flap was retracted anteriorly exposing the lateral surface of condylar head and the lower joint space. A condylotomy was now performed with a carbide burr at a level just below the widest circumference of the head of the condyle (Figs. 6 and 7). When this was completed, the fibers of the external pterygoid that attached to the anterior surface were dissected and the head of the condyle was removed (Fig. 8).

Fig. 4. Fig. 5.

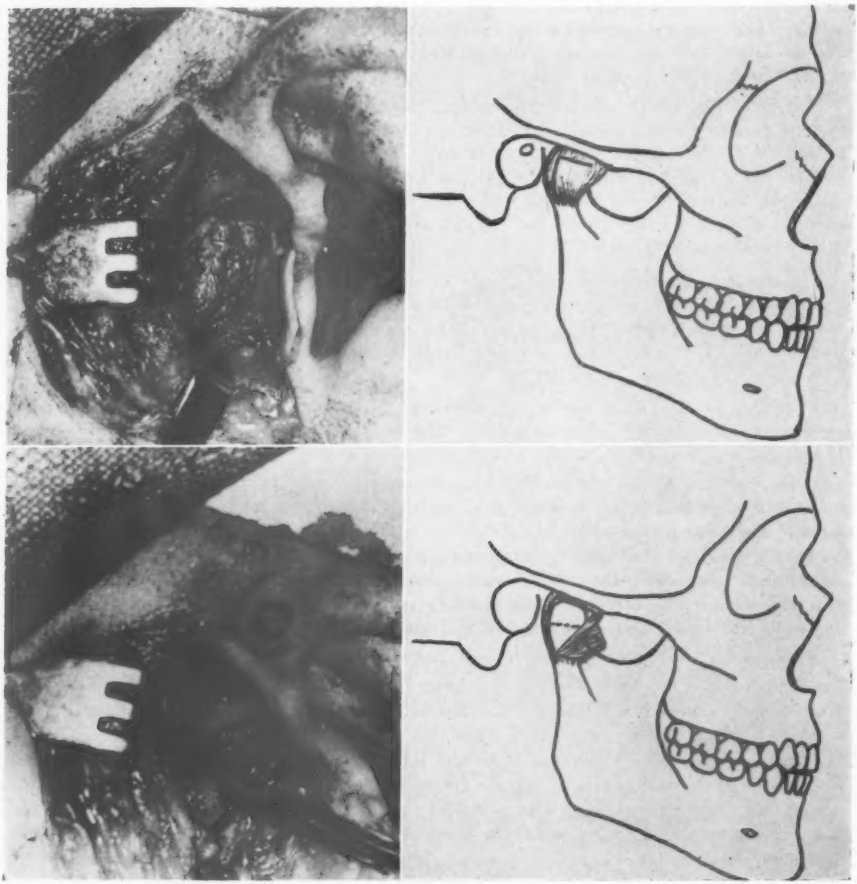


Fig. 6. Fig. 7.

Fig. 4.—Zygoma and capsule of joint are now visible.
Fig. 5.—The inverted "L" incision in the capsule.
Fig. 6.—A high condylotomy has been performed with a burr.
Fig. 7.—The cut below the widest circumference of the head of the condyle.

A small piece of Gel foam was placed in the wound to control a small amount of bleeding, and the capsule was closed with one plain catgut 000 suture. The wound was now closed in layers, using 000 catgut on the deeper layers and a 34 gauge stainless steel subcuticular

wire on the skin flap, with seven interrupted Dermalon 000000 sutures to approximate the skin margins (Fig. 9). Clean cotton was placed in the ear, and a dressing of Telfa covered by gauze pressure and tape was used to cover the wound. Intermaxillary elastic traction was now used to immobilize the jaws.

The patient's postoperative course was uneventful. There was no recurrence of severe pain or spasm in the joint area. Opiates were discontinued on the third postoperative day. The skin sutures were removed on the fourth postoperative morning, and the patient was discharged from the hospital.

Fig. 8.

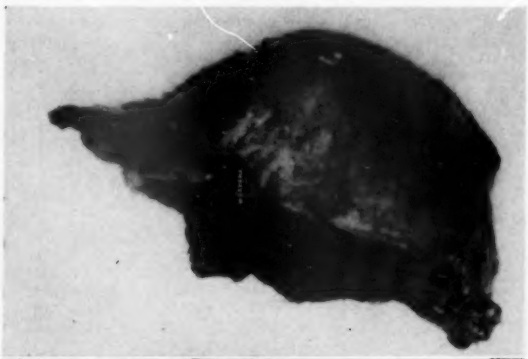


Fig. 9.

Fig. 8.—The removed head of the condyle with fibers of external pterygoid muscle.
Fig. 9.—Skin incision closed with subcuticular wire and skin sutures.

On the sixth postoperative day the intermaxillary elastic traction and wires were removed (Fig. 10). The patient was able to open her jaws 2.5 cm. with relative ease (Fig. 11). During the next few weeks the patient's ability to use and to open her jaws increased.

The patient returned to active nursing duty on the eighth postoperative day. The subcuticular wire was removed on the tenth postoperative day.

Two months following the surgery the patient was apprehended by narcotics agents as she was leaving the hospital from her shift as a floor nurse. Suspicion had been aroused by other nurses as to the possibility of her having taken some Demerol and codeine from the

Fig. 10.

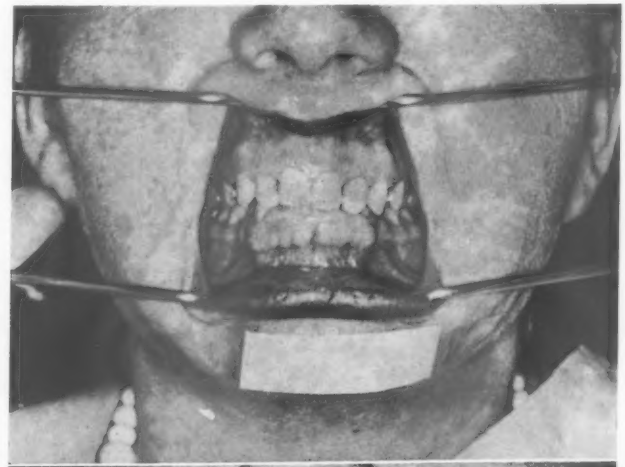


Fig. 11.

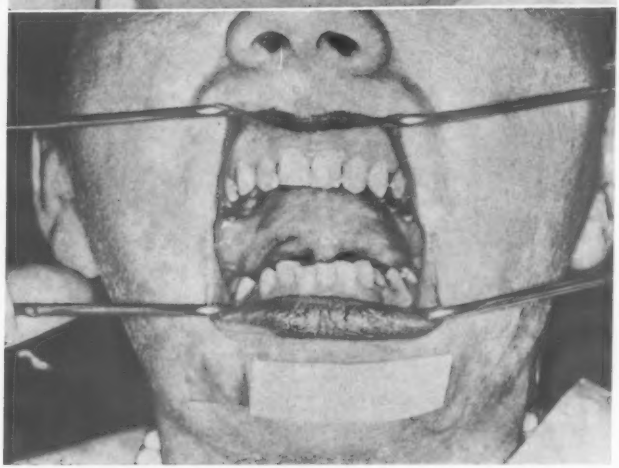


Fig. 10.—Patient's jaws in centric occlusion one week following surgery.
Fig. 11.—The patient one week postoperatively showing ability to open jaws 2.5 cm.

floor. The patient pleaded guilty in court and was given a suspended sentence when the judge was made familiar with her medical history. She was not truly addicted to the drugs but was well on her way toward drug addiction. Since that time she has taken no more of the narcotics.

The patient has been followed for the past fifteen months without recurrence of muscular spasm or dislocation. Her ability to use her jaws has increased 100 per cent. She now can open her jaws 5.0 cm., as compared to 2.5 cm. before surgery (Fig. 12). There is no discomfort in the joint, and there is only a 2 mm. deviation of the mandible to the left when the mouth is fully opened.

Fig. 12.—Fifteen months later the patient can open her jaws 5.0 cm.

Fig. 13.—Fifteen months postoperatively, the scar is well hidden.

There is no disturbance of the facial nerve, the scar is completely hidden (Fig. 13), and the patient can use her jaws with complete ease. The patient is now the mother of a baby girl.

DISCUSSION

This case shows what prolonged derangement of the temporomandibular joint may lead to in the way of distress to the patient. Perhaps earlier surgical treatment might have prevented the patient's lengthy period of distress with near addiction to opiates. This surgical procedure, which can be performed in one and one-fourth hours, helped to relieve pressure on the disc and relieve the reflex nerve pattern which caused spasms of the external pterygoid muscle with the resultant unilateral dislocation of the mandible.

SUMMARY

A case of prolonged, chronic, intermittent unilateral dislocation of the mandible has been presented. The final result was an acute prolonged spasm of the external pterygoid muscle on the affected side. Several types of treatment, including equilibration of occlusion, immobilization of the jaws, the use of sclerosing solutions, anti-inflammatory agents, muscle relaxants, and finally surgery, were employed. Complete relief was finally obtained following a high condylectomy.

REFERENCES

1. Sicher, Harry: Structural and Functional Basis for Disorders of the Temporomandibular Articulation, J. Oral Surg. 13: 275, 1955.
2. Thoma, Kurt H.: Oral Surgery, St. Louis, 1958, The C. V. Mosby Company.
3. Christensen, Robert W.: Surgical Correction of Complete Bilateral Ankylosis of the Mandible, ORAL SURG., ORAL MED. & ORAL PATH. 8: 1235, 1955.

90 NORTH OAKLAND AVE.

SOLITARY MYELOMA OF MANDIBLE

Report of a Case

R. I. H. Whitlock, M.D.S.(Lond.), F.D.S.R.C.S.(Eng.), and
N. C. Hughes, F.R.C.S.(Eng.), Belfast, Northern Ireland

ALTHOUGH cases of multiple myeloma with jaw lesions are fairly common in the literature, and a few such cases have been first diagnosed from the jaw lesion,[1-4] the solitary myeloma or plasmacytoma in the jaws is an extremely rare condition. As far as can be ascertained, only ten cases have been reported;[5-13] of these ten cases, six occurred in the mandible and four in the maxilla. It should be stressed, however, that lesions which show heavy infiltrations of plasma cells are fairly common in the oral region. Although most of these are inflammatory lesions which histologically can easily be differentiated from plasma-cell neoplasms, there may be difficulty in diagnosing a given section. Doubt can therefore be placed on the accuracy of the diagnosis of some of these reported cases, and it is quite possible that a few were not neoplastic but inflammatory in origin.

There are no apparent histologic criteria by which we are able to distinguish solitary from multiple myeloma. Consequently, there has been much argument as to whether or not the single lesion is a forerunner of the generalized type. Ewing[14] considered multiple myeloma a neoplasm characterized by multiple foci of origin and rarely metastasized. Others, including Cutler and associates,[15] believe that multiple myeloma may originate as a single lesion with subsequent metastases to other bones. Most authorities today believe in the multicentric origin of myeloma, but a single survey of the skeletal system is not sufficient to exclude it since its multicentric origin may not manifest itself for several years. Boyd,[16] however, states that the plasmacytoma is a rare, localized variant of the generalized type, that it is usually composed of well-differentiated plasma cells rather than highly neoplastic cells, and that it is amenable to treatment. Christopherson and Miller,[6] in reviewing ninety-four cases of the solitary type occurring in bone, also believe it to be a clinical entity, unrelated to multiple myeloma, with a favorable prognosis.

From a study of the reported cases of the solitary type of myeloma occurring in the jaws, notably those cases described by Christopherson and Miller,[6] Lane,[7] Davis and Havens,[10] and Hinds and Pleasants,[13] there does seem

From the Plastic and Maxillo-Facial Centre, Throne Hospital, Belfast, Northern Ireland.

to be some basis for this belief. Whereas multiple myeloma runs a natural course of from a few months to an average of two years after diagnosis, the solitary myeloma may pursue a benign course for several years with apparent cure. The following case of a supposedly solitary myeloma with no recurrence five years after operation gives further support to this view. Although perhaps it is not a distinct entity, such a tumor behaves clinically in quite a different way from the generalized type. In this respect, it bears some resemblance to the reticulum-cell sarcoma.

CASE REPORT

The patient, a 67-year-old retired farmer, was referred from another hospital in 1953 for diagnosis and treatment of a large, rounded, nontender swelling at the point of the chin.

History.—The patient had first noticed a swelling on the right side of his lower jaw some sixteen years earlier. This swelling had gradually increased in size over the years until it had now become most pronounced over the point of the chin. There was a history of an

Fig. 1.—Preoperative appearance showing swelling on chin.

operation on the jaw twelve years previously and another eight years previously. Both procedures had been performed via an intraoral approach. (An attempt made at the hospital to obtain some case notes regarding the operations was unsuccessful.) Recently the patient had become worried about his appearance, and he also had encountered some difficulty in eating since there was only one area on the right side of the mandible which was not tender when he chewed upon it.

There was nothing relevant in the past medical history; the patient had always enjoyed good health.

Examination.—

General: The patient was a cheerful, elderly-looking man with no cyanosis, anemia, or finger-clubbing. The physical examination revealed no abnormality.

Extraoral findings: A large, rounded, fluctuant but painless swelling about the size of a tennis ball was present at the point of the chin (Fig. 1). The overlying skin was thinned but of normal color and freely mobile. The lower border of the mandible could be

palpated on both sides from the angle to the premolar region, but the external surface of the bone on the right side felt expanded and uneven. There was no nerve palsy, anesthesia, or lymphadenopathy.

Intraoral findings: Twelve teeth were present: $\frac{3\ 2\ 1\ |\ 1\ 2\ 3\ 4\ 5\quad 7}{|\qquad\qquad\qquad\quad 6\ 7\ 8}$. The lower left first, second, and third molars were extremely loose. The lower alveolus was expanded both buccally and lingually from the $\overline{8}|$ region to the $|\overline{8}$ region. The mucous membrane had ulcerated, revealing the underlying growth, in three areas: the $\overline{8}|$ region; the $\overline{6\ 5}|$ region, where the growth had become pedunculated; and from the $\overline{2}|$ region to the $|\overline{6}$ region, where there was a large, oval-shaped, ulcerated area obliquely set (Fig. 2). No provisional clinical diagnosis was made.

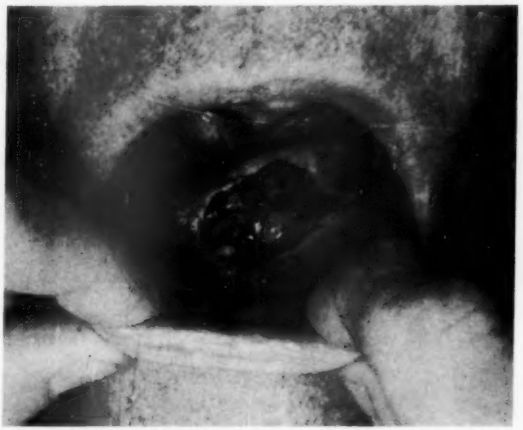


Fig. 2.—Ulceration in mouth.

Roentgenographic findings: There was a polycystic expansion of the mandible from the right ramus to the region of the lower left first molar (Fig. 3). At the symphysis (Fig. 4) there was a spherical shadow corresponding to the fluctuant area on the chin, across which traversed radiating spicules of bone in the form of a sunray.

A provisional diagnosis of adamantinoma, or endosteal osteoclastoma, was made. Osteogenic sarcoma was also considered but was ruled out because of the long history.

A biopsy was now considered. Because of the disfigurement, however, it was decided that a radical operation involving removal of the fluctuant growth at the point of the chin should be performed.

Operation.—Following induction of endotracheal anesthesia, two curved submandibular incisions were made to enclose the swelling over the chin. From their juncture they were extended on either side to the angles of the mandible. This part of the growth was found to be a soft solid, and it shelled out quite easily with the overlying skin attached. The rest of the growth was contained within the expanded body of the mandible; on both sides the outer plate was much thinned and had become deficient in several places. There was no expansion at the lower border, which was still intact. After the remainder of the outer plate had been peeled off with bone nibblers, the inside was found to be riddled with multiple cavities of varying sizes separated by incomplete bony septa; it contained a brownish fluid and was lined by what appeared to be an epithelial lining. The growth extended from the ramus on the right side to the premolar region on the left side.

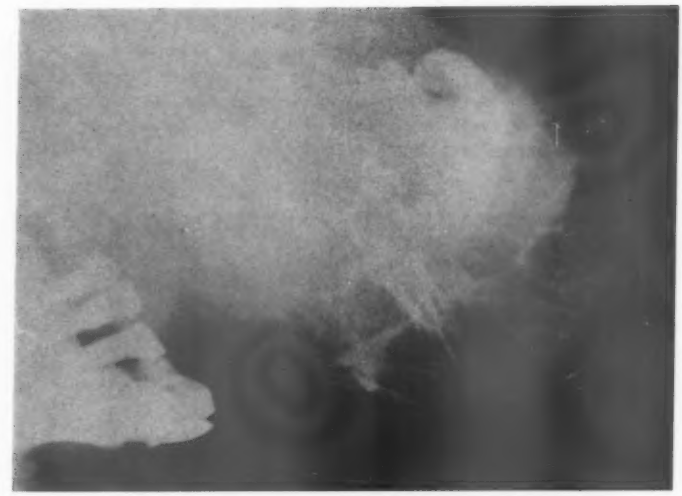


Fig. 4.—Soft-tissue lateral roentgenogram showing sunray appearance.

Fig. 3.—Roentgenogram showing cystic areas in left body of mandible.

On the basis of this clinical appearance, the lesion was assumed to be an adamantinoma. Because of its extent, however, a radical resection with an immediate bone-graft replacement was not feasible, and therefore a radical local removal was decided upon. All the cavities were carefully curetted, and the bone was trimmed back with bone nibblers until only the lingual plate remained. The lower left first, second, and third molars were then removed, and great care was taken in repairing the mucous membrane of the mouth, especially where it had been ulcerated. The external incision was next repaired in layers, and two corrugated-rubber drains were inserted extraorally. Healing was quite uneventful. The drains were removed after forty-eight hours, and in twelve days the patient was discharged from the hospital with the mouth and external incisions completely healed.

Pathologic Report (I. R. H. Kramer).—

Macroscopic: Two specimens were submitted. The first was a heart-shaped soft-tissue mass measuring 30 by 24 by 20 mm. The surface was brown and mainly smooth, and the cut surface appeared a "marbled" brown and white. The second specimen was a roughly spherical tumor, 6.5 cm. in diameter. The superficial surface was partly covered by a half-circle of skin 7 cm. in diameter; the deep surface was rough and contained embedded spicules of bone. When cut, the tumor was mainly firm and chocolate brown; it appeared to be bounded peripherally by a thin layer of bone.

Microscopic: The first tumor specimen was mainly composed of densely packed rounded and polygonal cells, each having a moderately bulky and sometimes "foamy" eosinophilic cytoplasm and a single large nucleus showing a very clear chromatin pattern (Fig. 5, *A* and *B*). There was some variation in nuclear size, and scattered throughout the tissue were cells with giant nuclei. The mass was moderately vascular and showed numerous areas of hemorrhage.

Peripherally, in some areas there was a fibrous capsule, but part of the surface was covered with necrotic tissue and fibrin infiltrated with polymorphs. Presumably, this area represented the site of oral ulceration.

Sections from the larger tumor mass (second specimen) showed essentially the same histologic appearances.

Diagnosis: Atypical myeloma.

Postoperative Observations.—Two months later (January, 1954) the patient was re-admitted to the hospital for further investigations in view of the possibility of his having generalized myeloma. A complete skeletal roentgenographic examination, however, failed to show any other lesions, and no further cavities had developed in the mandible. There was, unfortunately, a fracture at the symphysis region, but clinically only slight pathologic movement was elicited, with no displacement or pain. It was not known whether this fracture had occurred at the time of surgery or whether a pathologic fracture had taken place subsequent to the operation. There was a slight evening rise in temperature, and a slight trace of Bence-Jones proteose was found in the urine.

All hematologic tests were within normal limits. Hemoglobin was 85 per cent = 12.6 Gm.

A bone marrow report revealed that the marrow was active. Granulopoiesis was normal, and erythropoiesis was normoblastic in type. Occasional plasmacytes were seen but in insufficient numbers to substantiate a diagnosis of multiple myeloma.

A study by D. W. Neill of serum samples, including electrophoretic pattern, revealed that total serum proteins were 9.2 Gm. per cent, and a qualitative assessment of the electrophoretic distribution showed a raised gamma globulin with some increase in the beta globulin. This was consistent with the state of affairs found in generalized myeloma.

Electrophoresis, the migration of colloidal particles under the influence of an electric current, gives us a method of separating the various blood proteins which cannot be separated chemically or by other physical methods. The separation in this case was done by the horizontal paper strip technique described by Grassmann and colleagues[17] with minor modifications.[18] The densities of the dyed protein bands on the dried strips were evaluated by graphic plotting with a strip densitometer. The protein concentrations corresponding to the curve so obtained were derived by planimetric measurement.

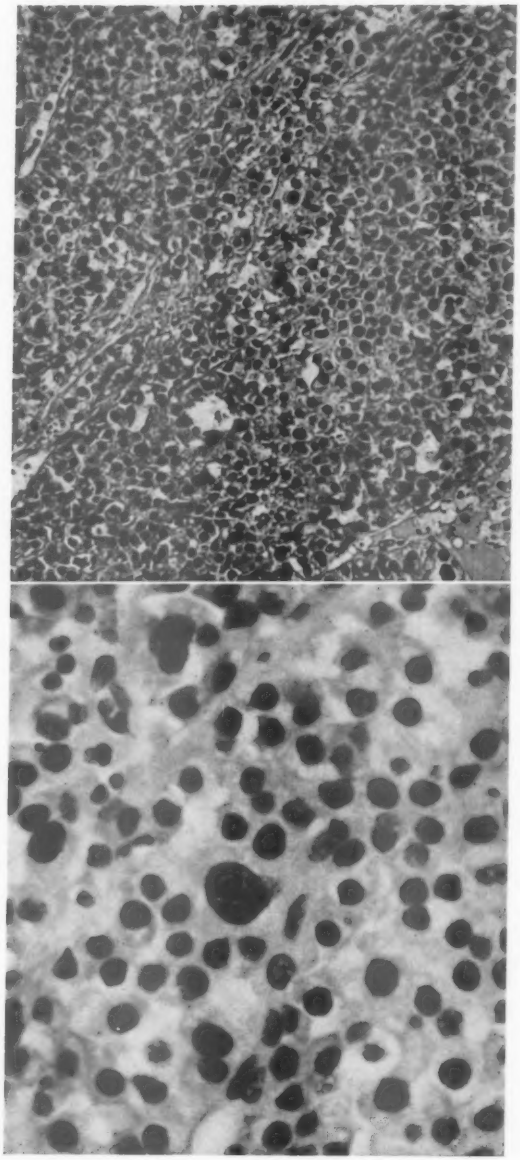


Fig. 5.—Photomicrographs of lesion. (Magnification: *A*, ×150; *B*, ×425.)

In April, 1955, sixteen months after the operation, the patient was again admitted to the hospital for a checkup. He had no complaints, and his general health had remained good during the intervening period. A complete skeletal roentgenographic examination was again negative, and this time the x-ray pictures of the mandible showed that bony regeneration had taken place in the cystic areas which had been curetted (Fig. 6). A fracture was still present in the symphysis region, however, but this was not troubling the patient. During this stay in the hospital the patient was fitted with a lower denture.

The hematologic examination was negative and this time there was no trace of Bence-Jones proteose in the urine.

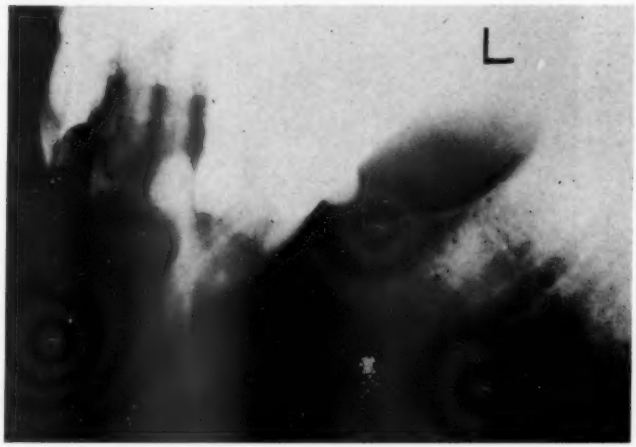


Fig. 6.—Lateral roentgenogram of left body of mandible two years after operation.

The total protein on this occasion was 7.4 Gm. per cent, and the contributions of the individual protein fractions were as follows:

Albumin	4.5 Gm. per cent
Alpha$_1$ globulin	0.2 Gm. per cent
Alpha$_2$ globulin	0.4 Gm. per cent
Beta globulin	0.7 Gm. per cent
Gamma globulin	1.6 Gm. per cent

This distribution was quite normal, the apparent slight rise in gamma globulin being well within the limits of experimental error of the method used.

Thus, after sixteen months the serum protein pattern had been restored to normal.

In February, 1957, three years and two months after the operation, the patient was again admitted to the hospital for investigations. His general health was still good, and he was even wearing his lower denture. Roentgenographic and hematologic examinations were negative, and there was no Bence-Jones proteose in the urine.

At this time the total protein was 7.4 Gm. per cent, with a distribution as follows:

Albumin	3.3 Gm. per cent
Alpha$_1$ globulin	0.5 Gm. per cent
Alpha$_2$ globulin	0.9 Gm. per cent
Beta globulin	0.9 Gm. per cent
Gamma globulin	1.8 Gm. per cent

While this pattern was not normal, the abnormality was a result of a fall in the albumin level with a corresponding rise in the globulin fraction. It would not appear to bear any relationship to the specific effect of plasmacytoma.

A further check was made in December, 1958. The patient was still well (Fig. 7). The serum study was reported as follows: "The pattern shows marked reduction in albumin with slight rise in gamma globulin plus marked rise in alpha$_2$ globulin. There is no suggestion of myelomatosis, but the pattern resembles that found in rheumatoid arthritis. There is definitely no indication of the rise in gamma-globulin which coincided with his plasmacytoma just after it was removed."

Fig. 7.—Appearance five years after operation.

DISCUSSION

The multiple myelomas are considered malignant tumors of the bone marrow in which the marrow cavity is expanded by a soft gray or red mass, but since there is no corresponding bone formation the cortex becomes thinned and pathologic fractures are common. The tumor, which varies from the size of a pea to the size of an orange, affects chiefly the sternum, ribs, and vertebrae with the skull, pelvis, and clavicle the next most frequently affected. The long bones are the least likely to be involved.

The tumor characteristically consists of plasma cells, but these do not show typical staining reactions and their nuclei are larger and do not have halos. Some pathologists have attempted to classify myeloma as to cell type (plasma, myeloma, etc.). Lichenstein and Jaffe,[19] however, state that the neoplasm has a single cell type. They say that this should not be subdivided into various subvarieties but that the cells, which may vary in their morphology, should be designated as the myeloma cells.

The disease, however, is not merely a bone marrow tumor; it is also characterized by abnormal protein in the blood, presumably produced by the enormous numbers of these "myeloma" tumor cells. This protein may act in different ways: (1) it may be excreted in the urine as Bence-Jones proteose (50 per cent of cases), and in advanced cases it may cause a blockage of the tubules

with subsequent renal failure; (2) it may be retained in the blood, causing a hyperproteinemia with a raised serum globulin, which probably accounts for the fact that in such cases there usually is only a trace of Bence-Jones proteose in the urine; or (3) it may possibly be deposited in the tissues as atypical amyloid. Due to the destruction of the marrow, anemia is also a symptom in a high proportion of cases.

Histologically, biochemically and electrophoretically, the tumor in the case reported here resembled the lesion seen in multiple myeloma. That the biochemical changes and the electrophoretic pattern were only mild is presumed to be due partly to the fact that the lesion was confined to the mandible and partly to the fact that tests were not performed until two months after the operation. It is interesting to note that in the case reported by Lane[7] a small localized plasmacytoma of the mandible caused an increase in the serum globulin ratio which began to return to normal two months after operation and was completely normal five months later.

The general belief now is that myelomas are multicentric in origin and that, although there is a possibility of a solitary lesion being first discovered, lesions in other bones very quickly follow. It might be argued that, inasmuch as our patient remained free from the disease five years after operation, there is a possibility that myelomas start as a local disease and early removal will result in a cure. In our case, however, there was only a thorough curettage instead of complete local removal, but this still resulted in complete healing. In this respect our case differs from that reported by Lane,[7] who performed a complete excision of the lesion, but in all other respects the cases are very similar.

As yet there is no means of differentiating plasma-cell tumors. These two cases, however, as well as the other nine reported in the literature, lend grounds for the belief that there occurs in the jaws a tumor which, although not a distinct clinical entity, has quite a different clinical course from generalized myeloma and is amenable to surgery. Such a tumor might be considered a solitary myeloma. Only time will tell, however, whether or not such a tumor will develop into one of the generalized variety.

SUMMARY

1. A case in which an extensive myeloma of the mandible healed despite the fact that only a curettage was performed is reported. The patient has remained well after five years, and so far generalized myeloma has not developed.

2. It is suggested that there is evidence to show that there occurs in the jaws a plasma-cell tumor which runs a different course from the generalized variety and is amenable to surgical treatment. This might be considered a solitary myeloma.

We wish to thank Mr. I. R. H. Kramer, M.D.S.(Lond.), Head of the Pathology Department, Eastman Dental Hospital, for pathologic reports and advice given in the preparation of this paper, and Mr. D. W. Neill, M.Sc., F.R.I.C., Principal Scientific Officer, Royal Victoria Hospital, Belfast, for the reports on the serum protein electrophoretic studies.

REFERENCES

1. Wolff, E., and Nolan, L.: Radiology **42**: 76, 1944.
2. Meloy, T. M., Jr., Gunter, J. H., and Sampson, D. A.: Am. J. Orthodontics & Oral Surg. **31**: 685, 1945.
3. Calman, H.: Oral Surg., Oral Med. & Oral Path. **5**: 1302, 1952.
4. Bruce, K. W., and Boyer, R. Q.: Oral Surg., Oral Med. & Oral Path. **6**: 729, 1953.
5. Spitzer, R., and Price, L. W.: Brit. M. J. **1**: 1027, 1948.
6. Christopherson, W. M., and Miller, A. J.: Cancer **3**: 240, 1950.
7. Lane, S. L.: Oral Surg., Oral Med. & Oral Path. **5**: 434, 1952.
8. Navarro-Sanche, F.: Odontoiatria (madr.) **9**: 105, 1952.
9. Mancusi-Materi, G.: Gior. ital. chir. **10**: 274, 1954.
10. Davis, G. D., and Havens, F. Z.: Proc. Staff Meet. Mayo Clin. **29**: 569, 1954.
11. Villa, V. G., and Laico, J. E.: Philippines Med.-Dent. J. **1**: 33, 1955.
12. Fink, K.: Medizinische **22**: 812, 1955.
13. Hinds, E. C., and Pleasants, J. E.: Oral Surg., Oral Med. & Oral Path. **9**: 193, 1956.
14. Ewing, J.: A Treatise on Tumours, ed. 3, Philadelphia, 1928 W. B. Saunders Company, p. 321.
15. Cutler, M., Busche, F., and Cantril, S. T.: Surg. Gynec. & Obst. **62**: 918, 1936.
16. Boyd, W.: Pathology for the Surgeon, ed. 7, Philadelphia, 1954, W. B. Saunders Company, p. 576.
17. Grassmann, W., Hannig, K., and Knedel, M.: Deutsche med. Wchnschr. **76**: 333, 1951.
18. Neily, R. A., and Neill, D. W.: Nature, London **176**: 33, 1955.
19. Lichenstein, L., and Jaffe, H. L.: A. M. A. Arch. Path. **44**: 207, 1947.

THE USE OF A TEMPORARY MAXILLOFACIAL PROSTHESIS IN SURGICAL-ONCOLOGIC CASES

Cicero B. Vianna, D.D.S., and Edmundo P. Fonseca, D.D.S.,**
São Paulo, Brazil

INTRODUCTION

THERE is no question but that plastic surgical repair following the extensive loss of facial tissue is far superior to a prosthetic appliance in the majority of instances. Unfortunately, however, there are often serious limitations to autoplastic repair, and in many instances a prosthesis is desirable pending plastic surgical restoration.

ADVANTAGES OF PROSTHETIC APPLIANCE

Actually, a prosthetic appliance may be desirable for a number of reasons: (1) the prosthetic therapy does not generally require hospitalization of the patient, (2) the area of surgery can be readily and periodically inspected,[1] (3) the appliance does not irrevocably contraindicate subsequent plastic repair at a later and more desirable time,[2] (4) a maxillofacial prosthetic appliance can be fabricated in a few days, (5) esthetic and functional effects are immediately possible, and (6) deficiencies of the appliance, such as discoloration and imperfect adaptation, may be easily corrected from time to time.

CASE REPORTS

Five cases are presented which were sent to us by the Head and Neck and Dental Services of the Hospital Central do Cancer de São Paulo. The time that elapsed between the removal of the neoplasm and the fabrication of the appliance varied from three to ten months. Consequently, the possibility of a recurrence of the neoplasm was constantly considered. This, in fact, is one of the advantages of a temporary appliance, since it permits ready inspection of the surgical zone.

CASE 1.—Fig. 1 shows the extent of the lesion and indicates the loss of the entire left orbit and proximal tissues. A strong scar resulted in immobilization of the left side of the lip. Fig. 1, *B* shows the appliance, and Fig. 1, *C* demonstrates the prosthesis in position.

From the Department of Maxillofacial Prosthesis, University of São Paulo School of Dentistry.

*Assistant Professor, Department of Maxillofacial Prosthesis, University of São Paulo School of Dentistry, São Paulo, Brazil (Chairman, Antonio de Souza Cunha, M.D., D.D.S.).

A. B.

C.

Fig. 1.—Case 1.

A. B.

C.

Fig. 2.—Case 2.

Fig. 3.—Case 3.

Fig. 4.—Case 4.

CASE 2.—This case has special significance since the appliance had to be made quickly so that the patient could return to his home at a distant point. Fig. 2, *A* demonstrates the extent of the lesion which included complete loss of the left orbit and of the neighboring tissues. Because of the speed with which the appliance had to be fabricated (four days), an ocular prosthesis was not made, as shown in Fig. 2, *B*. The appliance in position is shown in Fig. 2, *C*.

CASE 3.—This patient was undergoing surgical repair, as shown in Fig. 3, *A*. In order for him to return home, however, it was felt that a temporary appliance should be made (Fig. 3, *B*). The arrangement in position is shown in Fig. 3, *C*.

CASE 4.—The extent of the lesion, with complete loss of the left orbit, is shown in Fig. 4, *A*. The appliance was made quickly to expedite the patient's return home (Fig. 4, *B*). Fig. 4, *C* shows the appliance in position attached to the glasses frame.

A. *B.*

C.

Fig. 5.—Case 5.

CASE 5.—This case differs from the others because of the associated loss of facial tissue and palate (Fig. 5, *A*). The appliance is depicted in Fig. 5, *B*. The prosthesis in position is shown in Fig. 5, *C*.

DISCUSSION

In this report emphasis has been placed upon the utility of *temporary* appliances. We have developed and employed in these five cases our own techniques for the fabrication of *permanent* prostheses, which will be related in a subsequent report.

SUMMARY

Five cases in which temporary facial prostheses have been constructed for patients with extensive tissue loss due to cancer have been discussed.

The temporary facial prostheses were made of acrylic resin.

REFERENCES

1. Burgoyne, J. R.: Oral Cancer, Philadelphia, 1954, Lea & Febiger, pp. 209-231.
2. Sarnat, B. G., and Schour, I.: Cancer da face e da boca, Rio de Janeiro, 1956, Ed. Cientifica, pp. 264-268.

EXODONTIA

. . . . . .

"DRY SOCKET" AFTER MANDIBULAR ODONTECTOMY AND USE OF SOLUBLE TETRACYCLINE HYDROCHLORIDE

James F. Quinley, D.D.S., R. Quentin Royer, D.D.S., and Robert J. Gores, D.D.S., Rochester, Minn.

MANY different antibiotics have been placed in tooth sockets immediately after odontectomy for the purpose of preventing "dry socket." Reports in the literature and a résumé of clinical experiences tend to indicate that the use of such medications has reduced this morbid entity. When tetracycline hydrochloride (Achromycin) became available in soluble form, it was thought that the drug should be tested to determine its effectiveness in the prevention of postoperative osteitis or dry socket after removal of impacted mandibular third molars. Therefore, the present study was undertaken.

In 1943 Millhon and associates[1] carried out a study in which they evaluated the incidence of dry socket in wounds caused by the removal of lower third molars when sulfonamide drugs and other socket dressings were used prophylactically. The results of their study served as a basis for comparison of the results of our study (Table I).

METHOD OF STUDY

In a group of 367 patients, 522 impacted mandibular third molars were removed. In 510 sockets, 50 mg. or more of Achromycin (Table II) was placed just prior to closure of the wounds. Most patients who were to undergo odontectomy for removal of an impacted mandibular third molar during a selected time were so treated; the only exceptions were those patients for whom systemic antibiotic therapy was necessary.

General anesthesia produced by Pentothal sodium with nitrous oxide and oxygen was used for 341 patients, and local anesthesia produced by a 2 per cent solution of Lidocaine with Adrenalin 1:100,000 was used for 26 patients.

In general, the surgical technique consisted of uncovering the impacted tooth and its surrounding bone by preparation of a mucoperiosteal flap and

From the Section of Dentistry and Oral Surgery, Mayo Clinic and Mayo Foundation. The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

by removing the overlying bone by means of the mallet-and-chisel technique. The impacted teeth were sectioned by splitting with a chisel, and each portion of the tooth was removed individually. The wounds were débrided and the flaps were closed by suturing.

Achromycin tablets (50 mg. each) were placed in the sockets just prior to suturing. No Achromycin was placed in twelve sockets; one tablet (50 mg.) was placed in eighty-one sockets; two tablets (100 mg.) were placed in 374 sockets; three tablets (150 mg.) were placed in thirty-seven sockets; four tablets (200 mg.) were placed in sixteen sockets; five tablets (250 mg.) were placed in one socket; and six tablets (300 mg.) were placed in one socket in which an associated dentigerous cyst was present prior to operation. The tablets were crushed and broken after placement within the sockets.

The operation in each case was performed by one of three surgeons, and each surgeon made his own postoperative observations.

Both examination of the socket and the patient's complaints relative to the severity of postoperative pain were used to denote the occurrence of dry socket. If the complaint of pain exceeded the usual amount of soreness, the socket was probed to determine the exposure of bone within the alveolar socket. When these two factors were positive and an obtundent drug was necessary to relieve pain, the diagnosis of dry socket was made.

Dry socket developed in eight of the 522 sockets from which impacted third molars had been removed. These eight sockets were found in eight different patients from each of whom only one impacted molar was removed. Of the 522 impacted molars removed, only twelve were removed without the insertion of any Achromycin tablets into the alveolar sockets. Dry socket developed in four of these twelve sockets, however, whereas there were only four dry sockets in the remaining 510 sockets that had been treated with one or more tablets of Achromycin. Certainly no definite conclusion could be drawn from this, but there is no doubt as to its significance. Table II indicates the results of the study.

We can conclude from Table II that the rate of incidence of dry socket when one or more tablets of Achromycin were used was 0.78 per cent. When nothing was used, the rate of incidence was 33½ per cent. The latter finding is not statistically significant because of the small series. The study by Millhon and associates indicated a rate of incidence of dry socket of 16.8 per cent in a series of 304 extractions in which no medication was used. They found a rate of incidence of 2.8 per cent when they used 60 per cent sulfathiazole in a glycerin base in a series of 397 extractions.

Although the results in this series are extremely favorable with regard to incidence of dry socket, it should be noted that several patients experienced a foreign-body reaction. The foreign body proved to be unabsorbed Achromycin. The reaction manifested itself after several weeks when a hard, insoluble, black, tarry-looking substance sloughed from the wound. Tissue and material from the affected site or sites were removed surgically and sent to the pathologist, and in all cases a report similar to that which follows was

TABLE I. INCIDENCE OF DRY SOCKET AFTER REMOVAL OF IMPACTED LOWER THIRD MOLAR TEETH, ACCORDING TO AGE, FOLLOWING USE OF VARIOUS DRUGS, WITH A CONTROL*

AGE (YEARS)	CONTROL			MEDICATED AND UNMEDICATED GAUZE TREATMENT			STERILE PETROLATUM			SULFATHIAZOLE (60 PER CENT) IN PETROLATUM			SULFATHIAZOLE (60 PER CENT) IN GLYCERINE BASE		
	TOTAL EXTRACTIONS	DRY SOCKET		TOTAL EXTRACTIONS	DRY SOCKET		TOTAL EXTRACTIONS	DRY SOCKET		TOTAL EXTRACTIONS	DRY SOCKET		TOTAL EXTRACTIONS	DRY SOCKET	
		NO.	PER CENT		NO.	PER CENT		NO.	PER CENT		NO.	PER CENT		NO.	PER CENT
Less than 20	29	3	10.3	6	0	—	26	2	7.7	24	0	—	49	0	—
20-29	171	23	13.5	66	0	—	122	13	10.7	144	10	6.9	223	6	2.7
30-39	72	19	26.4	46	2	4.3	55	4	7.3	30	1	3.3	81	4	4.9
40-49	18	4	22.2	20	3	15.0	23	1	4.3	15	1	6.7	34	1	2.9
50-59	12	1	8.3	9	0	—	8	0	—	9	1	11.1	5	0	—
60-69	2	1	50.0	2	0	—	3	0	—	0	0	—	5	0	—
Total	304	51	16.8	149	5	3.4	237	20	8.4	222	13	5.9	397	11	2.8

*From Millhon, Austin, Stafne, and Gardner.[1]

TABLE II. INCIDENCE OF DRY SOCKET AFTER USE OF ACHROMYCIN

DEVELOPMENT OF DRY SOCKET	NUMBER OF SOCKETS AND DOSE OF ACHROMYCIN						
	NO ACHROMYCIN	50 MG.	100 MG.	150 MG.	200 MG.	250 MG.	300 MG.
Present	4	1	3	0	0	0	0
Absent	8	80	371	37	16	1	1
Total sockets	12	81	374	37	16	1	1

presented: vascular inflammatory granulation tissue showing foreign-body giant-cell reaction around amorphous pink-staining material (Figs. 1 and 2).

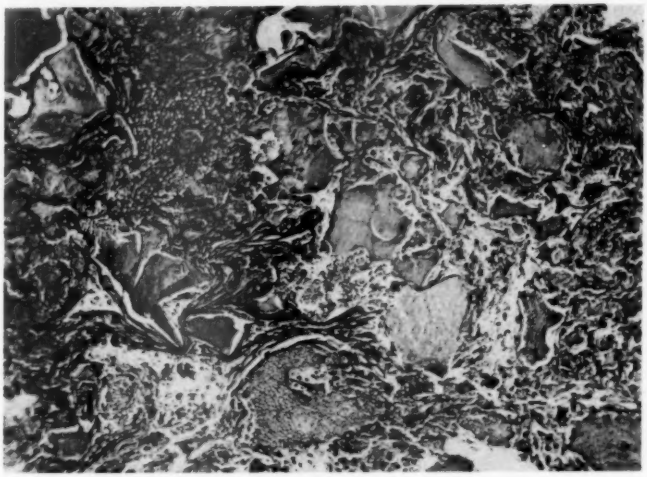


Fig. 1.—Low-power view of foreign-body substance. Note the amorphous pink-staining material. (Eosin stain. Magnification, ×110.)

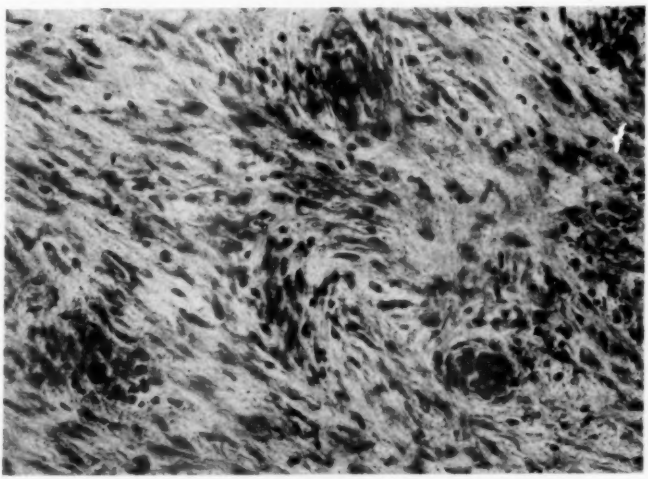


Fig. 2.—High-power view of foreign-body reaction. Note the giant cells. (Magnification, ×225.)

In order to see if this insoluble substance could be reproduced in vitro as it was seen in vivo, a specimen of it was sent to the biochemical laboratory for study. The following experiment was carried out: A 50 mg. tablet of Achromycin was sealed within a fresh beef heart for twenty-four hours. The

protein in the beef heart absorbed the free hydrochloric acid. The free base of the Achromycin went into the solution and then crystallized out as a black, insoluble substance.

The same reaction took place when a 50 mg. tablet of Achromycin was placed in 2 c.c. of serum. Crystals of insoluble free base occurred.

Both substances were in the same photospectrum as Achromycin when dissolved in tenth-normal hydrochloric acid.

The patients experienced little pain or discomfort as a result of this foreign-body reaction; however, it was annoying and time-consuming, both to the patient and to the surgeon.

In the majority of cases the use of Achromycin has been most satisfactory as shown by the incidence of dry socket, which proved to be less than 1 per cent with its use. It is imperative to note the foreign-body reaction in a small percentage of cases.

In conclusion, it is our opinion that the disagreeable results that naturally follow a careless surgical technique cannot be avoided merely by the use of chemotherapy or antibiotics.

REFERENCE

1. Millhon, J. A., Austin, L. T., Stafne, E. C., and Gardner, B. S.: An Evaluation of the Sulfa Drug and Other Dressings in "Dry Socket" in Lower Third Molars, J. Am. Dent. A. 30: 1839-1844, 1943.

PERIODONTICS

CURETTAGE BY ULTRASONIC INSTRUMENT

Preliminary Report

Henry M. Goldman, D.M.D., Boston, Mass.*

ONE of the basic procedures in the periodontal therapy armamentarium is curettage. The prefix "sub-" has been employed to denote that it is the sulcular wall where the débridement is performed. In this report the term "curettage" is defined as a curettement or débridement of the sulcular wall to remove that portion of the gingival wound constituting the surface of the diseased gingival attachment (the pocket) so that healing may occur. Hand instruments (curettes, for the most part) are used for the operation. The purpose of this report is to illustrate some findings of the curettage procedure with the use of an ultrasonic instrument.

PROTOCOL

Fifty adult patients varying in age from 36 to 65 years, with moderate to advanced periodontal disease denoted by pocket formation of not less than 3 mm., were selected. Health questionnaires were filled out, and those with any systemic manifestation were so recorded. Of the group, four patients had complications: one had chronic nephritis, one had rheumatic heart disease, and two had diabetes. Preoperative photographs were taken. Of the fifty patients, thirty-eight were subjected to biopsy to ascertain the status of the gingival tissue at selected times following instrumentation. The anterior portion of the dentition was preferred so that instrumentation would be more uniform.

For this investigation, various procedures were performed as follows: (1) immediate resection of an area scaled and curetted by the ultrasonic curette at the same time and of an untreated control area (eleven cases); (2) resection of cases similar to the preceding but allowed to heal for one week (eight cases); (3) resection of cases similar to those in the first group but allowed to heal for three weeks (five cases); (4) resection of an area débrided by the ultrasonic curette one week after the removal of calculus by hand instrument and of an untreated control area (seven cases); (5) resection of cases similar to those in the fourth procedure but allowed to heal for three weeks (seven cases).

*Professor of Stomatology, Boston University School of Medicine.

The excised gingival tissue was fixed in formalin and prepared for microscopic examination. The routine stain was hematoxylin and eosin. Several sections of each specimen were made to obtain a representative finding.

CLINICAL OBSERVATION

Fifty cases were observed clinically, as stated in the protocol. These were carefully examined for tissue tone, retraction, bleeding, and exudate. Repre-

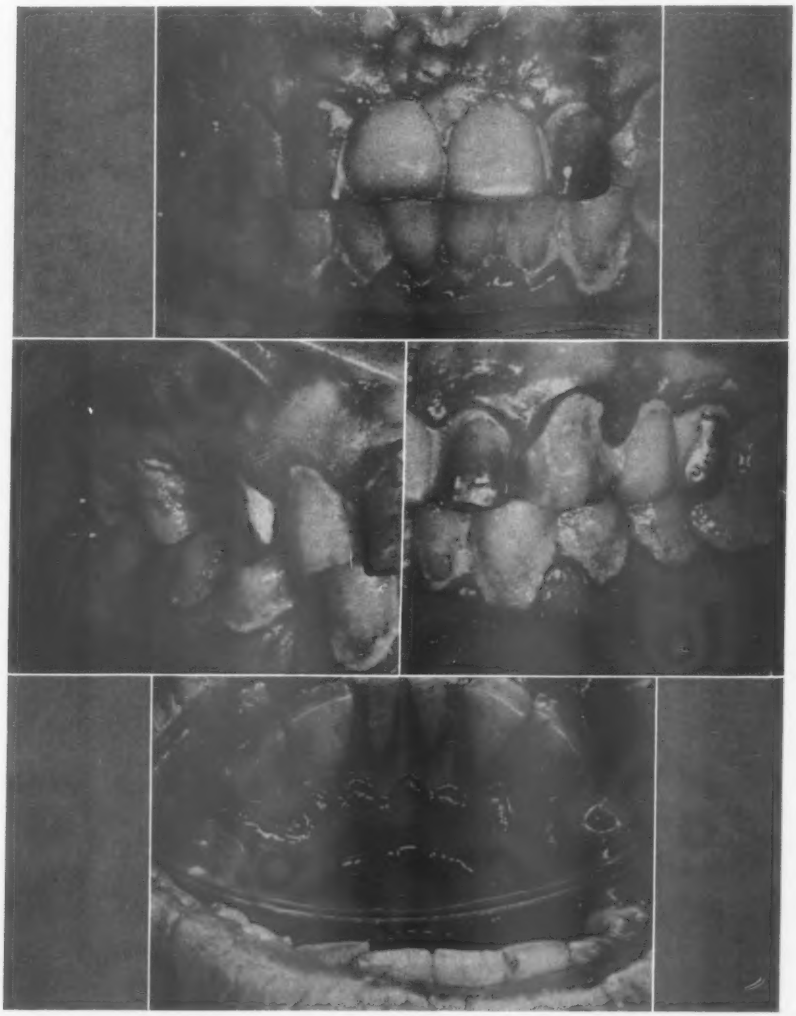


Fig. 1.—Photographs of the dentition of a patient with rheumatic heart disease. Not only were there very heavy calculus deposits throughout, but it was possible to express a purulent exudate by pressing against the gingivae. When the sulcular area was probed, bleeding occurred.

sentative of the successfully treated cases is one seen in Figs. 1 to 4. The patient, a 46-year-old man with rheumatic heart disease, presented an advanced periodontal manifestation characterized by gingival deformity, retraction, a marked purulent exudate, and bleeding upon probing. Because of the extensive and intense gingival disease, it was decided to premedicate the patient with

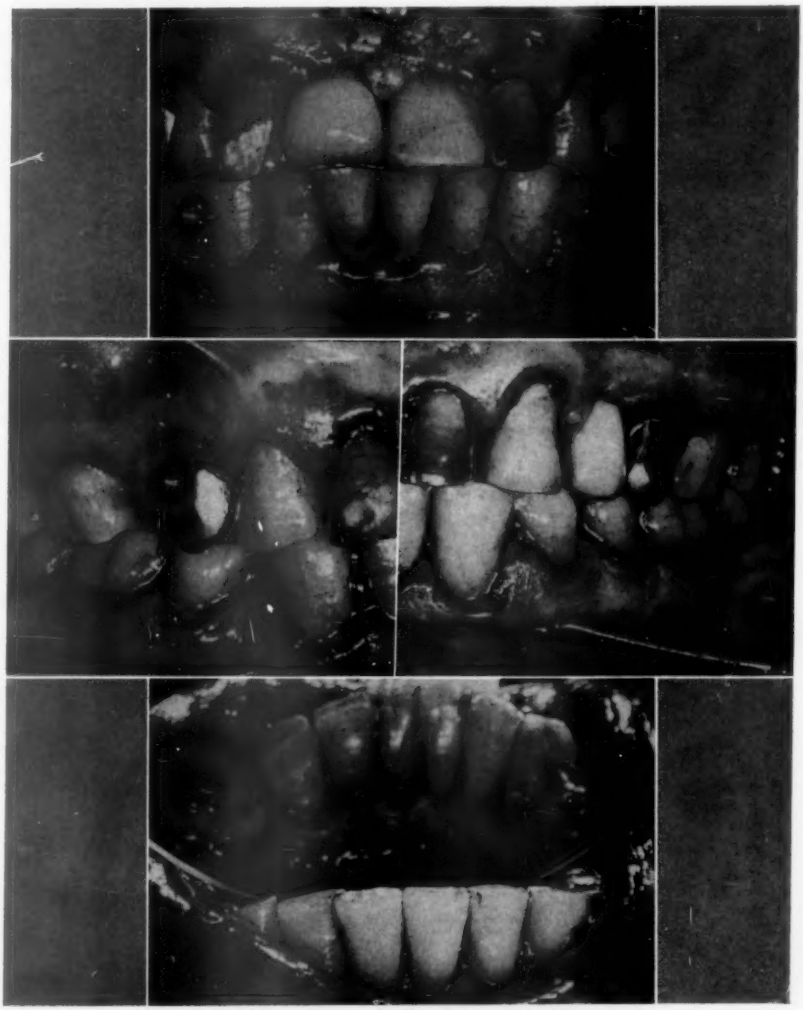


Fig. 2.—Photographs of the dentition of the patient seen in Fig. 1. Because of the systemic background, antibiotic therapy was instituted before, directly after, and for two days following treatment. The operative procedure was done in a single sitting. The retracted gingival tissue after débridement, with some oozing of blood, can be seen. In this instance not only was the débridement performed, but also the calculus was removed with an ultrasonic instrument.

penicillin and to scale and débride the gingival tissue with the ultrasonic instrument in one sitting. The supragingival heavy calculus was removed first, followed by attention to the sulcular wall. Photographs were taken directly after the procedure (Fig. 2). Postoperatively, penicillin was prescribed; there were no complications, and healing was uneventful. The photographs taken after one week are seen in Fig. 3. The patient was instructed in an oral hygiene program.

Fig. 3.—Photographs of the case seen in Figs. 1 and 2 one week after the curettage procedure. Note the change in color, form, and adherence of the gingival tissue as well as the shrinkage. The patient up to this time had not been taught an oral hygiene program but brushed indiscriminately as well as he could. The oral hygiene program was instituted at this sitting.

Fig. 4 shows photographs taken two months later during a routine checkup. In the meantime, a new maxillary anterior fixed bridge has been constructed. One may note the contour, texture, and form of the gingivae in these latter photographs and compare them to those seen in the ones taken before treatment. In this instance there was marked regression of the gingival inflammation. Postoperatively, the gingivae were firm, pink, and adherent to the teeth. Shrinkage had occurred. Comparison of the gingival margins in the "before" photograph to those in the "after" photograph reveals the change in form and contour that took place.

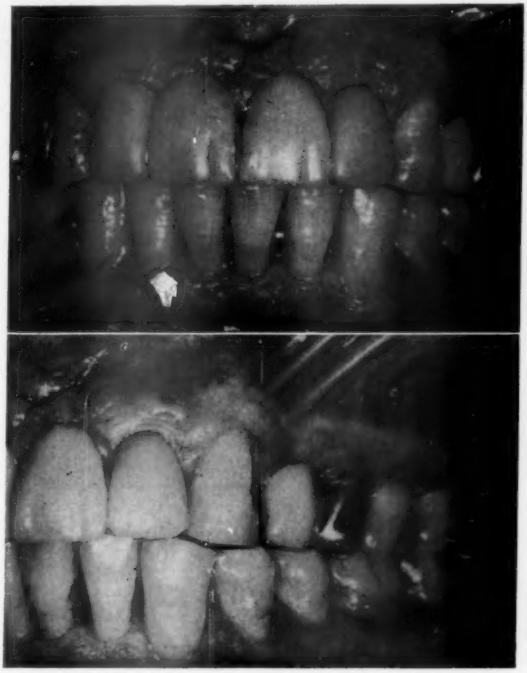


Fig. 4.—Photographs of the case seen in Figs. 1, 2, and 3 two months later. Note the character of the gingiva at this stage. The patient had been checked on his oral hygiene program. A new temporary anterior fixed bridge has been made.

In review of the cases studied, successful clinical results were obtained in almost all the cases. It is of interest, however, that despite a marked improvement in clinical appearance, histologic examination of removed tissue did not always disclose complete healing of the gingival wound. (Complete epithelization and repair in the gingival corium constitute complete healing.)

MICROSCOPIC FINDINGS

In the first series of cases, the gingival tissue was excised immediately following the curettage procedure. When these sections were compared with the

control sections, it was apparent that the sulcular wall had been débrided, so that for the most part no sulcular epithelium or epithelial attachment remained. Of interest was the fact that after débridement the sulcular wall was smooth and not "chewed up." The surface was that of the gingival corium in which there was the same degree of inflammatory infiltrate as that observed in the control specimen. In two of the nine specimens some small residue of epithelial rete pegs could be seen. In four of the sections a coagulum on the surface was present.

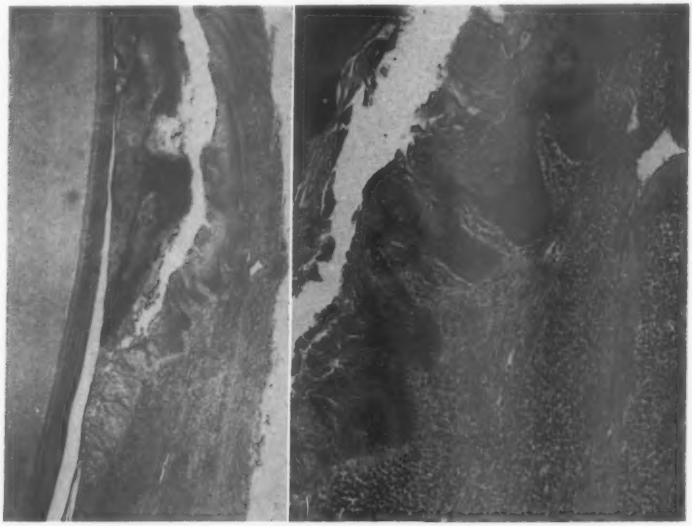


Fig. 5.—Low- and high-power photomicrographs of a pathologic gingival attachment (pocket) in which calculus is seen adherent to the tooth surface. The changes in the sulcular epithelium with keratinization and proliferation of the rete pegs and a dense inflammatory infiltrate in the corium are evident.

In the cases in which healing was allowed to take place for one week, it was found that epithelization was complete in three cases but not in five. In explanation, three possibilities exist: (1) that calculus removal had not been complete, (2) that débridement was deficient, and (3)that the time lapse was not sufficient. In the three cases in which wound healing was complete, it was found that there was a surprising lowering of the inflammatory infiltrate content of the gingival corium. Also, the collagen element was higher, denoting a healing of the gingival tissue. Those cases allowed to heal for three weeks were essentially the same as that described after one week, except that of the five cases, three showed complete epithelization.

Of extreme interest were the seven cases scaled prior to débridement with the ultrasonic curette and allowed to heal for one week. In these instances complete epithelization occurred over the sulcular area, with diminution of inflammatory infiltrate and collagen production in six of seven cases. In contrast, the control specimens showed all the characteristics of diseased gingival tissue.

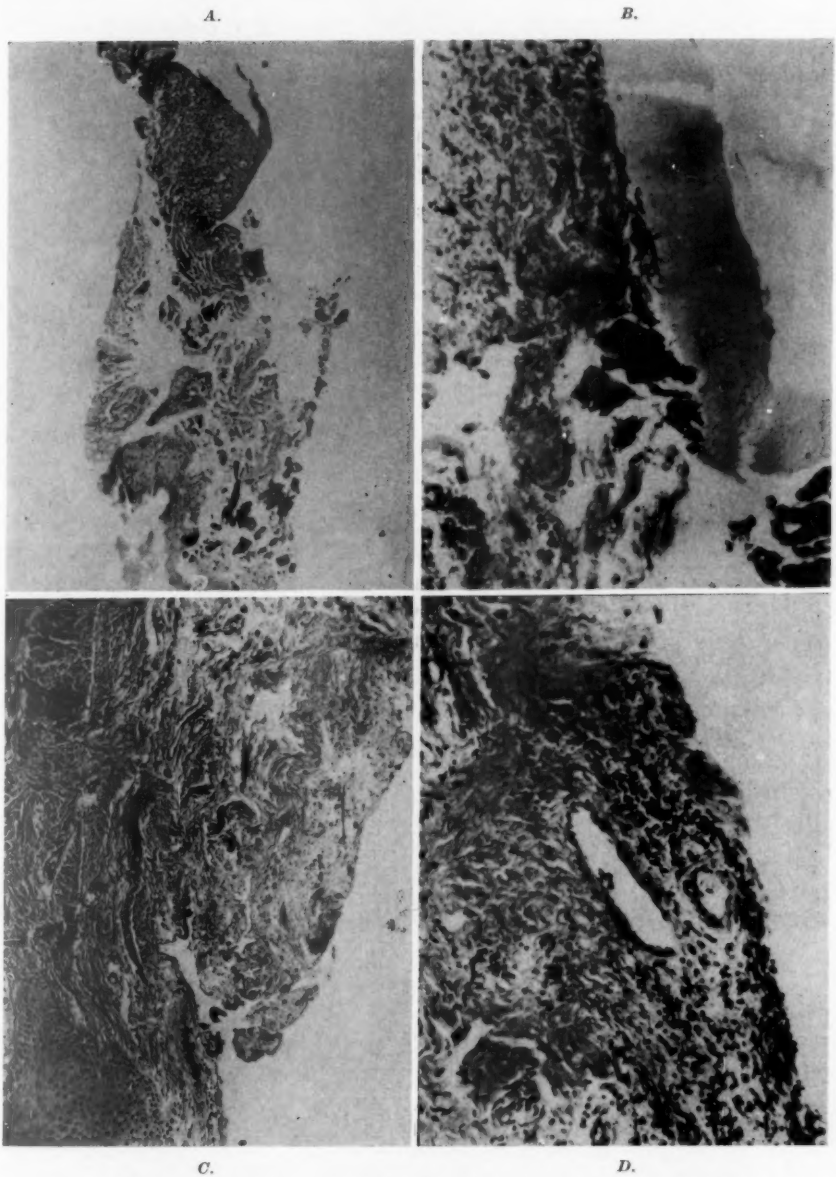


Fig. 6.—Photomicrographs of gingival biopsies following ultrasonic curettage. In *A* the contents of a pocket can be seen. It is composed of calcified material, inflammatory connective tissue, and epithelium. At *B*, on the curetted surface, a coagulum can be seen. *C* represents a low-power photomicrograph of the surface curetted by the ultrasonic instrument. Note the even surface completely denuded of epithelium. In *D*, a high-power view, a surface curetted by an ultrasonic instrument can be seen. The débrided surface is composed of a loose connective tissue in which there are numerous inflammatory cells.

These former specimens illustrated gingival repair as evidenced by complete epithelization of the sulcular wall and resolution of inflammation of the gingival corium. Of the seven cases in which biopsy study was made after three weeks,

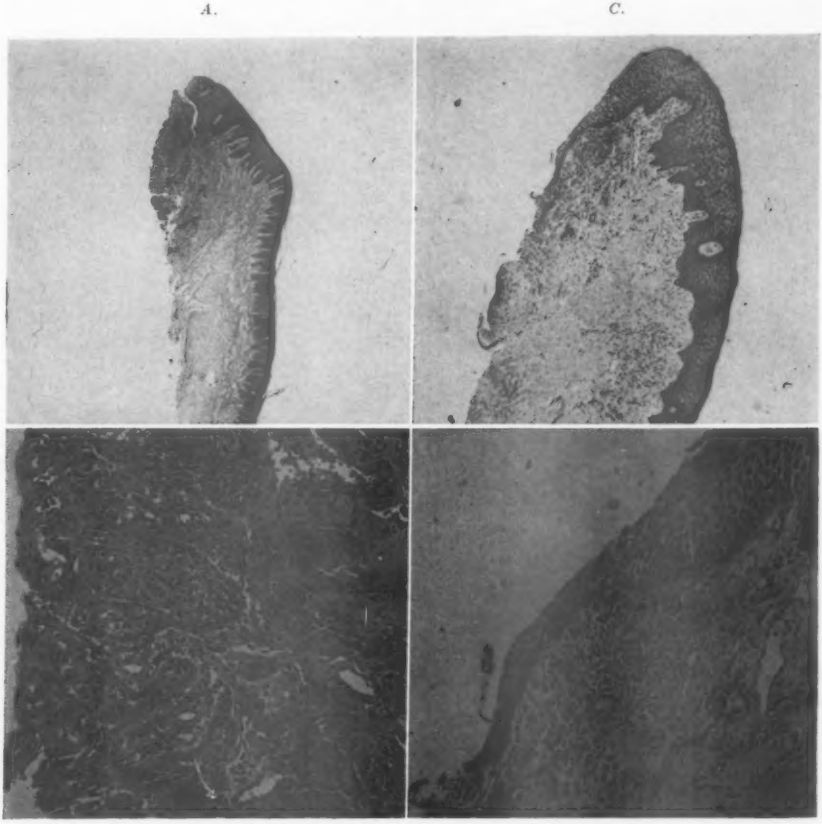


Fig. 7.—Low- and high-power photomicrographs of a control section (*A* and *B*) and an operated section (*C* and *D*). The control section shows all the characteristics of a diseased gingival attachment (pocket) with proliferation of the sulcular epithelium and a dense inflammatory infiltrate throughout the corium. Collagen fibers are undergoing breakdown near the sulcular wall, while there is some proliferation of collagen throughout the gingival tissue, per se. In the biopsy specimen of the treated area, the sulcular epithelium is intact. Subepithelially, there is an inflammatory infiltrate. Further away from the sulcular area there is a minimum of inflammatory cells with the corium consisting for the most part of dense collagen fibers. (From Group 2.)

five illustrated healing. In the remaining two cases it was apparent, in contrast to the controls, that some repair occurred but not to the extent of the others; in these, there was a distinct higher content of inflammatory infiltrate with some proliferation of the sulcular epithelium.

Fig. 10 is a composite drawing of the changes which seemingly resulted from analysis of the cases studied microscopically. In the left-hand drawing all the microscopic findings of pocket formation are apparent. Calculus can be

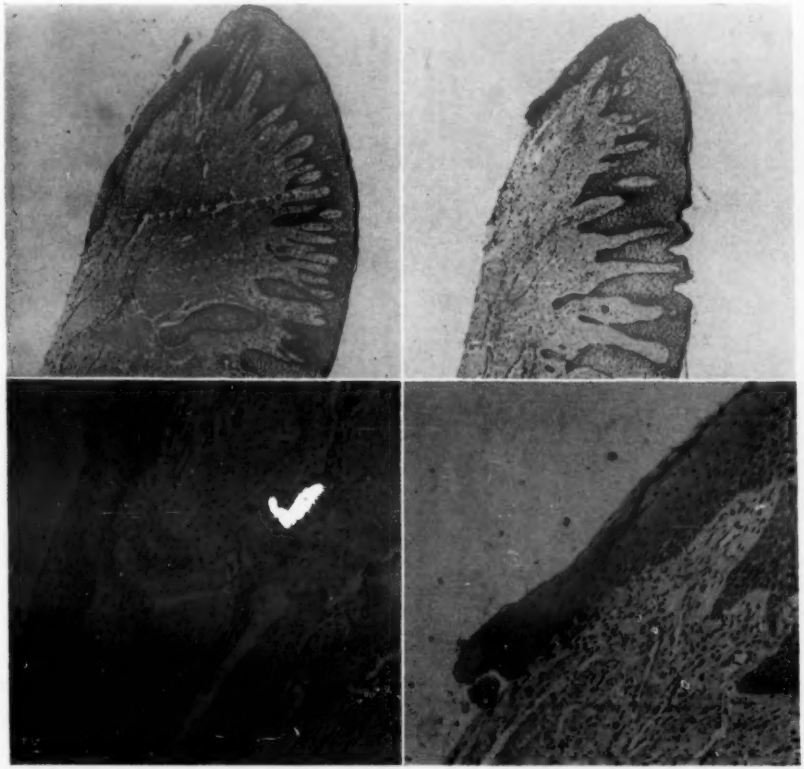
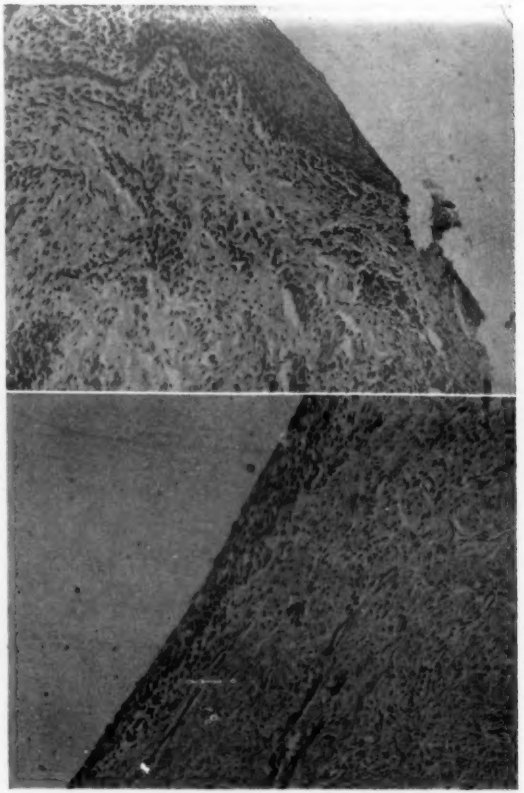


Fig. 8.



A.

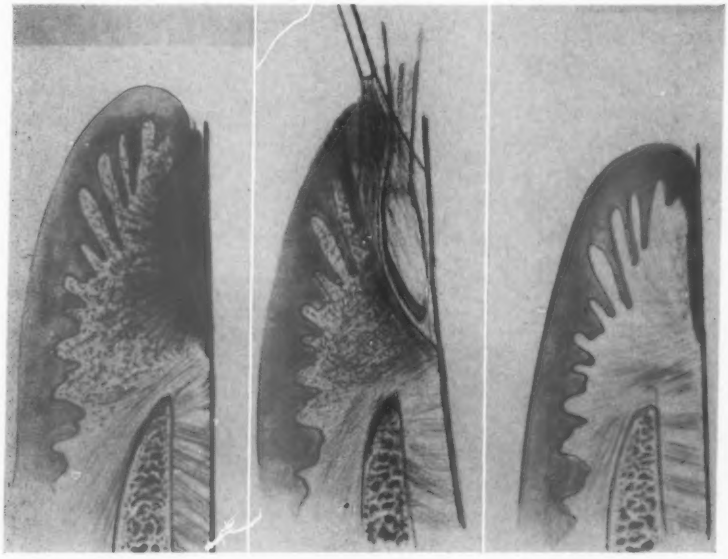
B.

Fig. 9.

Fig. 8.—Low- and high-power photomicrographs of two sections three weeks after curettage with the ultrasonic instrument. Note the intact sulcular epithelium and the character of the corium. (From Group 5.)

Fig. 9.—*A* and *B* represent high-power photomicrographs of the sulcular epithelium and corium of two gingival sections treated three weeks previous by ultrasonic débridement. (From Group 5.)

seen occupying the entire pocket area; it lies in direct apposition to the sulcular epithelium which is undergoing pseudoepitheliomatous hyperplasia. The rete pegs are proliferating into the gingival corium, with areas of ulceration exposing the subepithelial tissue to the surface. There is a distinct inflammatory infiltrate throughout the entire gingival tissue, but it is more concentrated near the sulcular area. The calculus has been removed, and in the next drawing the ultrasonic curette is shown performing the débriding operation. In the



A. *B.* *C.*

Fig. 10.—A composite drawing of the findings of ultrasonic débridement in this experiment. At *A* the characteristic diseased gingival attachment with calculus present in the pocket can be seen. *B* illustrates the débriding operation, and *C* shows the healing of the gingival tissue. Photomicrographs in the previous Figs. 7, 8, and 9 allow for a composite picture, making up part *C* of this figure. The length of the epithelial attachment has been taken by measurements of the entire length of the regrown sulcular epithelium. In some instances it was short, less than 1 mm.; in others for the most part it ranged from this measurement to a little over 2 mm. A few specimens were over this figure. It is difficult to ascertain whether or not this sulcular epithelium is attached to the tooth surface.

third drawing, healing has occurred. The epithelium is now intact and extends along the cementum from 1 to 2 mm. With resolution of inflammation, the inflammatory infiltrate is practically gone; in some specimens there was a localized infiltrate subepithelially. Collagen fibers occupy the gingival corium. From clinical observation, it appears that some degree of shrinkage has occurred.

The third drawing can be substantiated by Fig. 7, in which the sulcular epithelium has regrown covering the corium in contrast to the control, an untreated gingival pocket. Measurement of the sulcular epithelial strip is slightly more than 1 mm.

CONCLUSIONS

The ultrasonic curettement constitutes an excellent method of débridement of the sulcular wall of the periodontal pocket. Histologic examination of excised tissue after curettage discloses that healing occurs by epithelization of the sulcular surface and resolution of inflammation in the gingival corium. Successful ultrasonic curettage is obtained when the tooth surface is thoroughly cleansed and the sulcular wall is completely débrided.

REFERENCES

1. Barkann, Lillian: A Conservative Surgical Technic for the Eradication of a Pyorrhea Pocket, J. Am. Dent. A. **26**: 61, 1939.
2. Bell, D.: Indication and Contraindication for Subgingival Curettage in the Treatment of Paradentosis, J. Periodont. **8**: 55, 1937.
3. Beube, F.: A Study on Reattachment of the Supporting Structures of the Teeth, J. Periodont. **18**: 55, 1947.
4. Box, H. K.: Treatment of the Periodontal Pocket, Toronto, 1928, University of Toronto Press.
5. Chaikin, B. S.: Subgingival Curettage, J. Periodont. **25**: 240, 1954.
6. Goldman, H. M.: Subgingival Curettage, a Rationale, J. Periodont. **19**: 54, 1948.
7. Hirschfeld. L.: Subgingival Curettage in Periodontal Therapy, J. Am. Dent. A. **44**: 301, 1952.
8. Ingle, J.: Periodontal Curettement in the Premaxilla, J. Periodont. **23**: 143, 1952.
9. Morris, M. L.: The Removal of Pocket and Attachment Epithelium in Humans: A Histologic Study, J. Periodont. **25**: 7, 1954.
10. Ramfjord, S.: Reattachment in Periodontal Therapy, J. Am. Dent. A. **45**: 513, 1952.
11. Waerhaug, J.: Microscopic Demonstration of Tissue Reaction Incident to Removal of Subgingival Calculus, J. Periodont. **26**: 26, 1955.

ORAL MEDICINE

. . . . . .

MERCURIAL NECROSIS OF THE CHEEK

Report of a Case

Aziz A. Rahimo, D.D.S., and Walter W. Shimasaki, D.D.S.,***
San Francisco, Calif.

SINCE the abandonment of heavy metal therapy in favor of antibiotics in the treatment of syphilis and the rigid enforcement of laws governing safety regulations for heavy-metal workers, metallic stomatitis, poisoning, or intoxication has become a rarity. However, the common usage of mercurial diuretics at the present time has stimulated the medical profession to study and evaluate these complications.

Aside from the occasional overdosage of mercurial preparations from accidental or suicidal ingestion, the diuretics, which are used primarily for the treatment of congestive heart failure and edema of cardiac origin, are probably the most dangerous of the mercurial drugs in therapeutic dosages. Toxic manifestations attributable to the direct action of these drugs may cause varied idiosyncratic reactions. In addition to renal damage, the predominating toxic manifestation occurs in the gastrointestinal tract. The symptoms are typically those caused by mercury, namely, stomatitis, salivation, and hemorrhagic colitis.[1]

Patients afflicted with mercurial intoxication present a picture of general malaise, anorexia, gastrointestinal disturbances, diarrhea, lassitude, and anxiety about their physical condition.

The oral findings are quite striking, as pointed out by Major and Bononi,[2] Hill,[3] and Burket.[4] The mouth shows a complete lack of good oral hygiene. Speech is often hoarse, labored, and painful. The breath is foul, and the saliva is viscous and profuse with a metallic taste. A generalized periodontal condition exists, with gingival margins around the teeth of a reddish purple color. Furthermore, the gingiva is tender, bleeds easily, and may show a

From the San Francisco City and County Hospital and the Graduate Division, Department of Oral Surgery, College of Physicians and Surgeons, School of Dentistry.
*Chief Resident, Oral Surgery, San Francisco City and County Hospital.
**Assistant Resident, Oral Surgery, San Francisco City and County Hospital.

bluish line along the margin. Teeth are sometimes loose and may form indentations along the margin of the swollen tongue. Grayish white membranous lesions may appear on the alveolar mucosa, mucobuccal reflex, cheek, and tongue. Multiple ulcers may be present to form a typical picture of ulceromembranous stomatitis. In severe cases these lesions may form a necrotic ulcer exposing the underlying bone and give the appearance of a tumor. With superimposed infection, an osteomyelitis may develop, and pathologic fractures sometimes occur.

The oral manifestations of mercurial stomatitis, as Lentz[5] pointed out, depend on three factors: (1) the presence of mercury, (2) the local conditions, and (3) the bacterial infection.

CASE REPORT

In January, 1958, a 56-year-old unemployed Mexican man came to the outpatient oral surgery clinic of San Francisco County Hospital with the chief complaint of soreness of the cheek. His present illness dated back to June 1, 1957, when he was admitted to this hospital with a chief complaint of a sore jaw of five days' duration. The local physician who referred the patient made an admitting diagnosis of diphtheria, based on the

Fig. 1.—Lesion of right cheek as seen on the first outpatient visit.

appearance of three greenish white necrotic areas found in the mouth and pharynx. The patient was sent to an isolation ward, and a study for diphtheria was negative. It was found that the patient also had diabetes mellitus, congestive heart failure, and renal disease. He was then transferred to a medical ward, where he was evaluated for a possible fungus infection of the mouth and treated for the other systemic findings. During his hospital stay, the patient was placed on a low-salt diabetic diet and given 100 mg. digitalis daily. Cultures of the oral lesion produced normal flora. No mercurial medication was given during his stay in the hospital. Urine and blood studies were within normal limits. An eye, ear, nose, and throat consultation was obtained, but the findings were negative.

Dental consultation revealed poor oral hygiene, with several loose, periodontally involved teeth. These teeth were removed, and the extraction sites healed uneventfully. The grayish white lesions in the mouth were healing spontaneously. The patient was discharged from the hospital one week after admission and was followed by the outpatient medical service.

Seven months following discharge from the hospital, the patient came to the outpatient oral surgery clinic with similar lesions in the mouth. He stated that he felt weak, sick, and tired. His appetite had been poor for the past several weeks. The clinical

examination at this time revealed a well-developed, well-nourished white man in acute distress. Asymmetry of the face was noted, with the right cheek slightly swollen. Bilaterally, lymph nodes of the submaxillary and superior cervical chains were palpable and slightly tender. The voice sounded hoarse and strained, and the breath was extremely foul.

Intraoral examination revealed a grayish white necrotic lesion of the right cheek at the level of the occlusal plane (Fig. 1). It measured 1 cm. in diameter and was irregular in outline. The necrotic tissue in the center was soft but could not be separated from its base. Surrounding this lesion was a firm, indurated base approximately 2 cm. thick. The patient stated that he noticed the lesion and swelling three days prior to this visit. Another desquamated, reddish lesion, measuring 1.5 cm. in diameter, was noted in the lower left mucobuccal fold of the cuspid region. This had been present for one week. The oral cavity showed poor oral hygiene, with deposits of calculus and materia alba at the gingival margins of all the remaining teeth. A generalized periodontitis existed. Copious amounts of saliva of a ropy but clear appearance were noted. The salivary glands were nontender and not enlarged. The palate, tonsillar pillars, and floor of the mouth were negative. Oral temperature was 100.2° F. Dental, face, and skull roentgenograms revealed no significant findings.

Fig. 2.—Same lesion four days later. Superior extension engulfs the area where the biopsy was taken.

Course.—A biopsy of the lesion of the right cheek was taken immediately. Upon questioning, the patient related the history as given above. He also disclosed that he was being seen by the medical service of this hospital and was receiving biweekly injections of an unknown drug. Awaiting the result of the microscopic examination, the patient was discharged and instructed to return four days later.

The original hospital records were reviewed, with no significant findings. Upon reviewing the case with the medical resident, it was discovered that the patient had been receiving biweekly injections of Mercuhydrin, a mercurial diuretic, since his discharge from the hospital. It was also discovered, through contact with the physician who originally referred the patient to this hospital, that the patient had been taking Salyrgan, also a mercurial diuretic, for a period of six months prior to his first admission with a diagnosis of probable diphtheria. On the basis of this information, the diagnosis of mercurial intoxication was made.

When the patient returned for the second visit, the lesion on the right cheek had enlarged to twice the size seen on the first visit and had invaded the muscular tissue to the skin (Fig. 2). The necrotic tissue was sloughing and becoming stringy. Areas of the lower right alveolar mucosa and tongue which contacted this lesion were also beginning to develop gray-white coatings (Figs. 3 and 4). The lesion of the lower left mucobuccal reflex had progressed. A histopathologic report disclosed an acute inflammatory reaction.

Treatment consisted of substitution of a nonmercurial diuretic, débridement of the necrotic tissue, painting the lesions with 2 per cent gentian violet, and an intensive course to improve oral hygiene. The patient was followed on biweekly visits for a period of one month. At the end of this time the oral lesions were well healed (Figs. 5 and 6).

Fig. 3.—The alveolar ridge contacting the lesion is becoming necrotic. Note the lack of oral hygiene.

Fig. 4.—Lesion now present on the tongue. This lesion also was in contact with the lesion of the cheek.

Fig. 5.

Fig. 6.

Figs. 5 and 6.—Appearance four weeks after initiation of treatment.

DISCUSSION

The case report just presented shows an unusual lesion of mercurial stomatitis with necrosis of the cheek. It is apparent that early diagnosis of such a lesion is extremely important. The discontinuation or replacement of the mercury-containing drugs and the maintenance of good oral hygiene constitute the main treatment in early cases.

The presence of mercury in the mouth of such patients may be localized in the gingival tissue or in the mucobuccal fold. Inflammation or swelling may subsequently be initiated in these regions due to local allergic, toxic, or irritative factors of the mercury. When good oral hygiene is lacking, the inflammatory areas may become bacterially involved and ulcerate, and necrosis may be augmented. The latter reaction, if not treated, usually progresses to attack all the adjacent soft and hard tissues.

Cardiac patients on mercurial diuretics should be advised to maintain good oral hygiene. Frequent visits to the dentist to eliminate early foci of infection or ulcerations of the oral cavity should be emphasized.

REFERENCES

1. Krantz, J. C., Jr., and Carr, C. J.: The Pharmacologic Principles of Medical Practice, ed. 2, Baltimore, 1951, Williams & Wilkins Company, pp. 954-955.
2. Major, G. S., and Bononi, S.: Osteomyelitis of the Jaws Following Acute Mercury Poisoning, Am. J. Orthodontics & Oral Surg. **25**: 83, 1939.
3. Hill, T. J.: Textbook of Oral Pathology, ed. 3, Philadelphia, 1945, Lea & Febiger, p. 352.
4. Burket, L. W.: Oral Medicine, ed. 2, Philadelphia, 1952, J. B. Lippincott Company, p. 78.
5. Prinz, H., and Greenbaum, S.: Diseases of the Mouth and Their Treatment, ed. 2, Philadelphia, 1939, Lea & Febiger, p. 214.

PHARMACOLOGY AND THERAPEUTICS

. . . . . .

DORIDEN: A QUIETING AGENT FOR ANXIETY IN ORAL SURGERY

William B. Linenberg, D.D.S., James H. Dirlam, Major, USAF (DC),** and H. James Harpole, D.D.S.,*** Atlanta, Ga.*

ACUTE apprehension and nervousness are probably the most common emotional problems encountered in patients undergoing oral surgical procedures. These conditions prevent a satisfactory doctor-patient relationship and hinder skillful performance of oral operations.

Until recently the barbiturates, with all the attendant risks of respiratory, circulatory, and central nervous system depression, toxicity, and habituation, were the only recourse in dentistry as well as in medicine for the control of apprehension and nervousness. In an attempt to find a more effective premedication for oral surgical procedures, we have evaluated a new nonbarbiturate sedative, glutethimide (Doriden†).

CHEMISTRY AND PHARMACOLOGY

Glutethimide (alpha-ethyl-alpha-phenylglutarimide) is a nonbarbiturate sedative compound which is quick acting and of short duration. It usually quiets tense, anxious patients without impairing psychomotor performance. This compound is a white crystalline powder which is relatively insoluble in water but soluble in alcohol and in acetone. The molecular weight is 217.3, and the melting point is between 83° and 85° C. Its action on the central nervous system is considered to be different from that of phenobarbital, but electroencephalographic tracings of the sleep pattern are similar to those seen with secobarbital and chloral hydrate.[1] There is no analgesic effect, even with large doses.[2] Body metabolism is not changed, oxygen consumption is not decreased, and there is no lowering of body temperature. Effects on the cardiac and respiratory systems are minimal, even with large doses; there is no hypotension or respiratory depression with therapeutic doses.[2]

*Assistant Resident, Department of Oral Surgery, Grady Memorial Hospital.

**Resident, Department of Oral Surgery, Grady Memorial Hospital. The contents of this article are the personal views of the author and are not to be construed as statements of official Air Force policy nor as Air Force endorsement of any commercial product described.

***Chief, Department of Oral Surgery, Grady Memorial Hospital.

†Doriden was supplied by CIBA Pharmaceutical Products, Inc., Summit, New Jersey.

The action of glutethimide administered by mouth begins in five to twenty minutes, the maximum effect being obtained in approximately thirty minutes. The duration of action is four to six hours.

METHOD OF STUDY

Seventy-nine patients, both male and female, with acute apprehension and nervousness were selected from the outpatient oral surgery service of Grady Memorial Hospital. Patients ranged in age from 15 to 79 years, with almost one-half of the group included in the 20- to 40-year age range. No attempt was made to select patients according to physical status, sex, or operation to be performed. Most of the operations included extractions, removal of impactions, and alveolectomies.

In order to evaluate these patients as objectively as possible, blood pressure and pulse values were obtained prior to the administration of 0.5 Gm. glutethimide. Since the maximum effect is obtained in thirty minutes, each patient was observed during this period. At the end of this time blood pressure and pulse rates were taken and the degree of sedation was noted before and during the operation.

For the purpose of tabulating the data, the results were divided into three categories:

1. *Good:* Patient was calm and relaxed with no perceptible nervousness or apprehension.
2. *Fair:* Patient was calm and cooperative, but there was perceptible nervousness or apprehension.
3. *Poor:* No sedative effect.

A placebo was also used to determine the psychological effect that it would have on a nervous and apprehensive patient.

RESULTS AND DISCUSSION

Of the seventy-nine patients studied, sixty-six received glutethimide and thirteen received a placebo. Of the thirteen that received the placebo, four were given glutethimide when it was found impractical to operate without some actual sedation.

Of the seventy patients receiving glutethimide, the sedative effect was "good" for oral surgical procedures in sixty-two cases (88 per cent). In seven patients (10 per cent), although the operation was performed, sedation was only "fair." In one case (2 per cent), sedation was "poor," and the surgical procedure was carried out with great difficulty.

In only one instance was the use of placebo effective in calming and relaxing the patient.

Table I shows a comparison of the blood pressure and pulse rates recorded before and after 0.5 Gm. glutethimide was administered.

The average systolic and diastolic decreases in blood pressure were 14 mm. Hg and 7.3 mm. Hg, respectively. The average decrease in pulse rate

was 10 beats. This decrease in blood pressure and pulse rate coincided with the observed degree of sedation. An increase in blood pressure and pulse rate at the same time was not observed in any patient. Any increase in blood pressure or pulse was negligible.

TABLE I. BLOOD PRESSURE AND PULSE RATES IN PATIENTS RECEIVING 0.5 GM. GLUTETHIMIDE

NUMBER OF PATIENTS	PER CENT	BLOOD PRESSURE	PULSE
39	56	Decrease	Decrease
8	11	Decrease	Increase
6	8	Decrease	Unchanged
9	13	Unchanged	Decrease
2	4	Unchanged	Unchanged
6	8	Increase	Decrease
70	100		

All patients were carefully questioned and observed in the clinic for evidence of dizziness, drowsiness, nausea, skin rashes, headache, and visual disturbances. Of the seventy patients receiving glutethimide, nine had a feeling of drowsiness, two complained of being dizzy, and one patient was both dizzy and drowsy. There were no other significant side reactions or postoperative sequelae.

To the present time, we have encountered no allergic reactions. Allergic reactions, which have been few, have been recorded by Howell[3] and Grabe.[4] They reported that the exanthema disappeared when the drug was discontinued.

Several fatal cases due to massive overdosage are reported by McBay and Katsas.[5]

Since several of the patients appeared quite drowsy following the oral surgical procedure, each was given 10 mg. of methylphenidate hydrochloride (Ritalin hydrochloride*). Methylphenidate hydrochloride is a mild stimulant and antidepressant which restores mental and physical activities to normal or near-normal levels, usually without producing hyperexcitability or depressive rebound. Within fifteen minutes all the patients were alert and were allowed to leave the clinic with another adult, whom they were previously instructed to bring with them.

SUMMARY

1. Alpha-ethyl-alpha-phenylglutarimide (Doriden), a nonbarbiturate sedative, was administered to seventy of seventy-nine apprehensive and nervous patients as a premedication for oral surgical procedures.

2. In sixty-two of the seventy patients 0.5 Gm. of glutethimide was effective as a sedative.

3. The maximum effect was obtained in thirty minutes.

*Ritalin hydrochloride was supplied by CIBA Pharmaceutical Products, Inc., Summit, New Jersey.

4. Drowsiness and dizziness were the only side effects encountered, and these were satisfactorily treated with 10 mg. of Ritalin administered orally. The effects of Ritalin were observed within fifteen minutes.

CONCLUSIONS

Glutethimide (Doriden) affords an effective and safe premedication for nervous and apprehensive patients about to undergo oral surgical procedures. As a premedicant, it demonstrates markedly good sedation and high safety and reduces the fears, anxieties, and apprehensive tendencies of most dental patients. For this reason, it is ideally suited as an antianxiety, tranquilizing agent in lieu of barbiturates or other premedicant drugs.

REFERENCES

1. Ladwig, H. A.: Electroencephalographic Study of Doriden, Arch. Neurol. & Psychiat. **74:** 351-355, 1955.
2. Gross, F., Tripod, J., and Meier, R.: Zur pharmakologischen Charakterisierung des Schlafmittels Doriden, Schweiz. med. Wchnschr. **85:** 305-309, 1955.
3. Howell, H. C. A.: Doriden, Brit. M. J. **1:** 399, 1956.
4. Grabe, G.: Kinderklinische Erfahrungen mit einem Glutarsaureimid (Doriden) als Schlaf und Beruhigungsmittel, Med. Klin. **50:** 1949, 1955.
5. McBay, A. J., and Katsas, G. G.: Glutethimide Poisoning: Report of Four Cases, New England J. Med. **256:** 97-100, 1957.

ORAL ROENTGENOLOGY

American Academy of Oral Roentgenology

Arthur H. Wuehrmann, Editor

BILATERAL DENS IN DENTE

George A. Morgan, D.D.S., F.A.C.D., and H. Guy Poyton, F.D.S., H.D.D., Toronto, Ontario*

DENS IN DENTE has been reported to be among the rarest developmental anomalies. Its occurrence in bilateral form has been recorded by few observers. In 1855 the *Transactions* of the Pathological Society of London carried what is now claimed to be the original article by Salter of the first dens in dente. Rushton was among the first to describe accurately this unusual type of dental developmental anomaly.

This epithelial invagination, which may originate in either the crown or the root of a tooth, has been tagged with various names, beginning with Salter's[1] "warty tooth." Busch,[2] in 1897, suggested the name of "dens en dents"; Rushton,[3] supported by Hunter,[4] suggested the name of "dilated composite odontome." Oehlers[5] prefers the more descriptive term "dens invaginatus."

Oehlers, in his papers on the subject, divides the various types of invaginations into three groups: (1) enamel-lined and confined within the crown; (2) enamel-lined in the form of a blind sac but root-invasive; (3) enamel-lined plus invasion and penetration of the root structure.

The name "dens in dente," meaning tooth within a tooth, was first suggested in 1897 by Busch, who believed that this phenomenon resulted from one tooth surrounding another through the process of growth. This presupposes that two tooth germs are involved, a concept that is no longer accepted by most investigators.

Present-day theory suggests that this particular condition results from an invagination of a single enamel organ. There is, however, continuing controversy as to the modus operandi of the invagination process. Rushton believed that it is a rapid and abnormal growth of cells of the deep surface of the enamel organ resulting in invagination of the dentine papilla. This growth

From the Department of Radiodontics, Faculty of Dentistry, University of Toronto.
*Fellow, American Academy of Oral Roentgenology.

process "presses the incompletely calcified dentine walls and papilla outwards so they are thinned and the deeper part of the bony crypt becomes enlarged. The part already calcified cannot be expanded; so in cases where the pressure

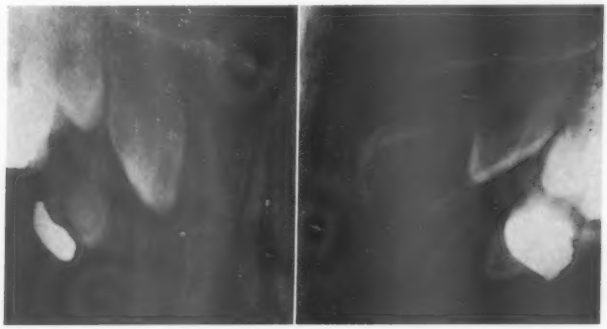


Fig. 1.—Case 1.

Fig. 2.—Case 1.

is great, the tooth becomes rapidly wider from the cusp rootward. The central pressure must force the crown in a superficial direction and may help to cause its conical shape."

Two cases of bilateral dens in dente are presented and illustrated.

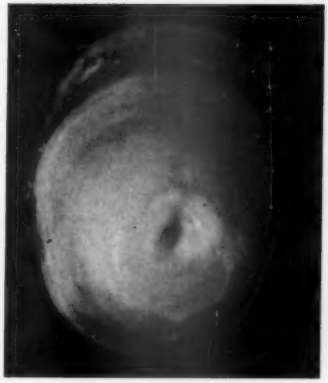


Fig. 3.—Case 1.

Fig. 4.—Case 1.

Fig. 5.—Case 1.

CASE 1.—The patient was a teenage girl with bilateral dens in dente located in the maxillary lateral regions (Fig. 1). Fig. 2 shows the clinical appearance of an extracted right dens in dente, and Fig. 3 demonstrates the clinical appearance of the incisal coronal portions of the extracted right dens in dente (note opening through crown).

Fig. 4 shows a roentgenogram of the removed right dens in dente. A photomicroscopic section of the right dens in dente is shown in Fig. 5; note that the enamel of both teeth is clearly illustrated.

CASE 2.—The patient, a teenage girl, had bilateral dens in dente in the maxillary lateral regions (Fig. 6). Both of these dens in dente have large areas of rarefying osteitis about their apices.

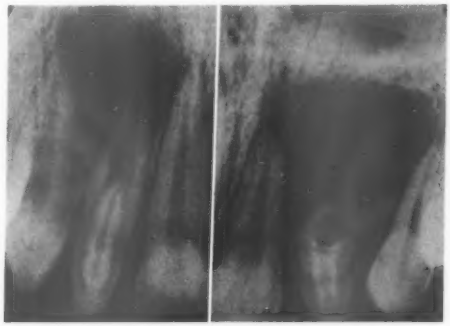


Fig. 6.—Case 2.

REFERENCES

1. Salter, S. J. A.: Warty Tooth, Tr. Path. Soc. London 6: 173-177, 1855.
2. Busch, F.: Über Verschmelzung und Verwachsung—Der Zahne des Milchgebisses und des bleibenden Gebisses, Deutsche Monatschr. Zahnh. 15: 469-486, 1897.
3. Rushton, M. A.: A Collection of Dilated Composite Odontomes, Brit. D. J. 63: 65-86, 1937.
4. Hunter, H. A.: Dilated Composite Odontome, ORAL SURG., ORAL MED. & ORAL PATH. 4: 668-673, 1951.
5. Oehlers, F. A. C.: Dens Invaginatus, ORAL SURG., ORAL MED. & ORAL PATH. 10: 1204-1218, 1302-1316, 1957. Radicular Variety of Dens Invaginatus, ORAL SURG., ORAL MED. & ORAL PATH. 11: 1251-1260, 1958.

ROENTGENO-QUESTIONS

How much reduction in the amount of radiation to the patient can be expected when aluminum filtration is added to the primary beam?

Each dental x-ray tube has some inherent filtration in the tube housing. Filtration in the form of aluminum disks 0.25 mm. or 0.5 mm. thick is usually added. Most recommendations suggest a total of inherent and added filtration of 2 mm., measured in aluminum or aluminum equivalents for kilovoltages up to 70. For kilovoltages in the 70 to 90 range, 2 to 3 mm., or even 3.5 mm., of filtration is suggested. When the above amounts are used, reductions from 27 to 57 per cent can be expected in the calculated skin dose.

How much reduction in radiation to the patient can be expected when the primary beam is collimated?

When the beam of primary radiation is reduced, so that at the outer end of the cone it is 2¾ inches in diameter, there is a marked reduction in the number of square inches of tissue radiated. A beam of this size is adequately large to cover the dental film with radiation during exposure and leave an adequate margin for small miscalculations in tube alignment without leaving areas of the film unexposed. Reducing the area radiated disproportionately decreases the amount of radiation administered to the patient. This disproportionately reduces the amount of secondary radiation generated which, in turn, reduces the fogging of the film and, therefore, improves the visibility of the details of the images on the film (tissue differentiation). When the diameter of the primary beam of radiation measured at the face is reduced from 3.5 inches to 2.75 inches, a 38 per cent reduction in the amount of radiation to the skin of the face is obtained. Some x-ray units come from the manufacturer with the diameter of the primary beam at the face greater than 3.5 inches. The percentage of reduction in radiation administered from these machines would be much greater than indicated in the example above if the size of the primary beam was reduced to 2.75 inches.

Collimation of the primary beam is usually accomplished by the use of a lead diaphragm with an aperture of the proper size in it, determined according to the cone distance used.

The American Academy of Oral Roentgenology solicits from the readers of ORAL SURGERY, ORAL MEDICINE AND ORAL PATHOLOGY any questions which deal with technical or interpretive aspects of oral roentgenology. These questions should be sent directly to the editor of this section, Dr. Arthur H. Wuehrmann, School of Dentistry, University of Alabama Medical Center, Birmingham, Alabama; the editor will distribute them for reply to a suitable group within the Academy. Questions, with the answers, will be published at as early a date as possible in "Roentgeno-Questions." Questions, especially those of an interpretive nature, should ordinarily be accompanied by good-quality roentgenograms and a pertinent history. The names of the questioner and the respondent will not be published.

ROENTGENO-ODDITIES

The Roentgenographic Appearance of Restorative Materials

The accompanying roentgenogram provides an interesting study in the roentgenographic appearance of dental restorative materials. Before reading further, the reader is encouraged to evaluate his knowledge of such matters by listing the restorative materials seen in this film.

The crown of the maxillary central tooth is covered by a metallic restoration. Its size and smoothness of contour suggest that the material is gold, but

---

Each month this section will bring to the readers of ORAL SURGERY, ORAL MEDICINE AND ORAL PATHOLOGY one or more roentgenograms which demonstrate unusual, unexpected, rare, or bizarre roentgenographic changes. These roentgenograms will be accompanied by an explanation or by words of inquiry, regarding the particular change. All films used in this section will be accepted with the understanding that 2 by 2 or 3¼ by 4 inch slides or glossy photographs of the films will be made available to interested readers through the editor of the American Academy of Oral Roentgenology at the individual's expense. The cost will involve only the photographer's charges. Each slide or print will give credit to the donor. Interesting roentgenograms are solicited from all sources. Please be certain to identify your films properly so that they can be returned. All material for publication should be submitted to Dr. Arthur H. Wuehrmann, University of Alabama School of Dentistry, Medical Center, Birmingham, Alabama.

the opaque qualities would be the same for silver, amalgam, or platinum. No line of cementation can be seen.

The cuspid, on its mesial surface, shows a circular invagination which, because of its shape, is much more likely to be a cavity preparation than caries. A slight opacity at the incisal portion of the preparation and, to a lesser degree, along the axial wall indicates the presence of a base, probably a zinc oxyphosphate base. Zinc oxide and eugenol would give a similar roentgenographic appearance, but because of its consistency when soft it is difficult to insert in as thin a layer as is shown. The circular space mesial to the base could be (1) a void (that is, restorative material missing), (2) a silicate cement, or (3) a plastic. Clinically, a silicate cement was present in reasonably good condition. The possibility of either a void or a plastic restoration is remote since both offer negligible resistance to x-rays and would look blacker than this area appears on the roentgenogram. The distal aspect of the cuspid shows a metallic restoration which clinically was determined to be silver amalgam. This portion of the roentgenogram is sufficiently distorted that interpretation is unwarranted.

The lateral tooth demonstrates several types of restorative materials. Gutta-percha is seen in the root canal; it appears to almost occlude the apex, but it lacks complete condensation throughout. The apical appearance is typical of a healed apicoectomy following endodontic therapy. A baked porcelain jacket crown covers an acrylic core which was processed onto a metallic post. The jacket is cemented to place with zinc oxyphosphate cement. The fact that acrylic is easily penetrated by x-radiation is demonstrated by the small, almost square, roentgenolucency in the corner of the coronal portion of the metallic post. In spite of the superimposed porcelain, this square area of acrylic is quite dark.

All of the above comments are substantiated clinically.

Arthur H. Wuehrman,
University of Alabama.

ORAL PATHOLOGY

STUDIES ON MINERALIZED DENTAL TISSUES

XIV. The Incremental Pattern of the Dentine in a Case of Osteogenesis Imperfecta

Gunnar Bergman, Odont. D., Stockholm, Sweden

THE histologic structure of the teeth in osteogenesis imperfecta has been described by several investigators.[1, 2, 4, 7, 8, 10, 15] Most authors agree that there is an exaggerated incremental lamination of the dentine in this condition and that the enamel has a normal structure.

My purpose in presenting this article is not to repeat well-known facts concerning the dental structures in osteogenesis imperfecta but to report some observations on the incremental growth pattern of the dentine of a patient who was under dental observation for thirteen years.

The incremental pattern of teeth in general has been compared with the formation of tree rings, and Westin[14] observed rhythmic changes in the dentine which led him to speak of "summer dentine" and "winter dentine." Schour and Massler[12] state: "The growing tooth acts as a kymograph which records within the structure of its growing enamel and dentin the normal and pathologic variations in metabolism."

CASE REPORT

The patient was a boy who had been admitted to the hospital several times since the age of 1 month with a diagnosis of osteogenesis imperfecta. He was frequently treated for fractures, the majority of them involving the femur. By the time the patient had reached the age of 13 years, twenty-three fractures had occurred. The history revealed no hereditary details of interest. The boy did not have blue sclerae.

From the time he was 12 months old, the patient was under dental observation at least once a year for thirteen years. Tooth eruption was not delayed, but on eruption the teeth had an abnormal bluish gray to yellowish gray color. The enamel of the deciduous teeth had a pronounced tendency to fracture, and there was also a tendency toward abrasion. One crown (02+) had been lost as the result of a transverse fracture at the neck of the tooth.

From the Department of Dental Histopathology, Royal School of Dentistry; the Department of Medical Physics, Karolinska Institutet; and the Eastman Dental Institute, Stockholm, Sweden.

The caries frequency was low, and the carious lesions were of an unusual type with no actual cavity formation but with superficial softening of the dentine in areas in which there were enamel fractures and abrasion. Roentgenographic examination disclosed that retarded dentinal development was common to all the deciduous teeth at an early age. When the patient was 18 months old, the anterior mandibular teeth had an abnormally thin dentinal layer and wide pulp chambers (Fig. 1, *A*) reminiscent of the "shell teeth" described by Rushton.[9] The deciduous molars showed a similar condition when the patient was 2½ years old. When he was 4 years of age, a rather small but definite increase in thickness of the dentine of the deciduous teeth could be observed (Fig. 1, *B*).

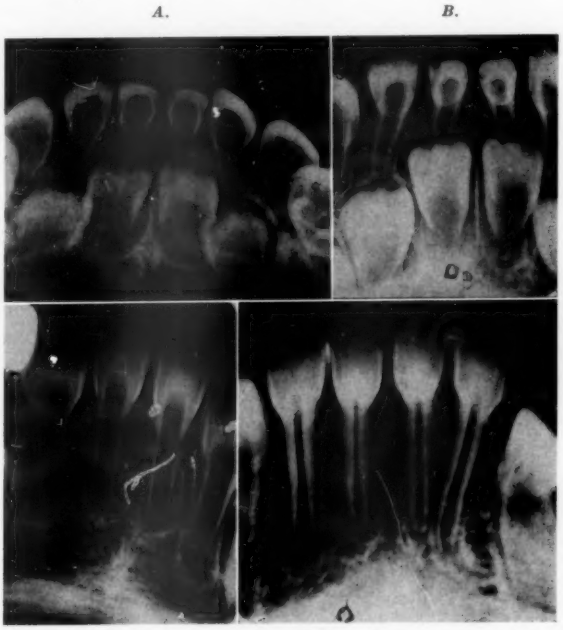


Fig. 1.—The anterior teeth in a case of osteogenesis imperfecta.
A, At 18 months, the dentine of the deciduous teeth is thinner than usual.
B, At 4 years, the dentine of the deciduous teeth has increased in thickness.
C, At 6½ years, the deciduous teeth have been shed. The dentine of the permanent incisors is thin in the roots but not unusually thin in the crowns.
D, At 10 years, the root dentine has increased in thickness and there is a tendency toward obliteration of the coronal pulp.

There was abnormally thin dentine in the roots of the permanent incisors (Fig. 1, *C*) and molars during the early stages of development, but the incisors differed from the molars by virtue of the marked growth of their coronal dentine. When the boy was 10 years old, there was a marked inclination to obliteration of the pulp chambers of the lower incisors (Fig. 1, *D*), but the permanent first molars at the same age did not show a similar tendency.

MATERIAL AND METHOD FOR HISTOLOGIC EXAMINATION

Five teeth were available for histologic examination: (1) the upper second deciduous molar (+05), extracted when the patient was 4 years old; (2 and 3)

the upper deciduous canines (+03, 03+), extracted when the patient was 10; (4) the lower second deciduous molar (–05), extracted when the patient was 11; and (5) the lower first permanent molar (–6), extracted when the patient was 14 years of age.

The teeth were fixed in formalin. The first permanent molar was split, and one half was ground to a thickness of 200 microns. The rest of the material was decalcified and embedded in paraffin. The 6-micron sections were stained with Mayer's hemalum-eosin and Bock, Hansen, and azan stains. The ground section was microradiographed according to the method of Engström and Wegstedt[5] with the equipment which I described in a 1957 publication.[3]

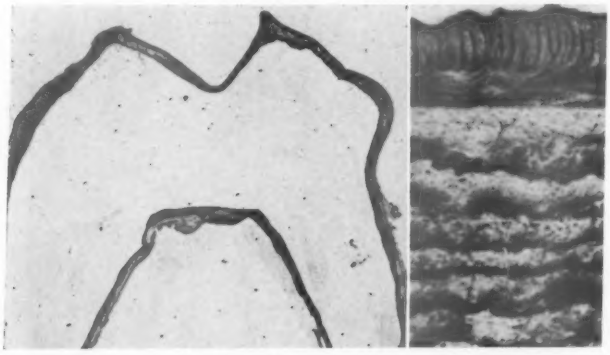


Fig. 2. Fig. 3.

Fig. 2.—Decalcified section of the upper second deciduous molar (+05) at 4 years, showing the abnormally thin dentine. (Bock's stain. Magnification, ×8; reduced ⅙.)

Fig. 3.—Detail from the section shown in Fig. 2. A layer of "mantle dentine" with regular structure is seen at the top of the photomicrograph. This layer and the adjacent dark band without visible dentinal tubules were formed before birth. The postnatal dentine in the lower part of the picture has a lamellar structure and few tubules. Hansen's stain. Magnification, ×275; reduced ⅙.)

OBSERVATIONS

The *enamel* of the first permanent molar was normal on histologic and microradiographic examination. No other enamel was available for microscopic examination since it was lost on decalcification. The *dentine* of all five teeth exhibited changes typical of osteogenesis imperfecta. The examined teeth differed only in respect to the thickness of the dentine; the youngest tooth had the thinnest dentine. Common to all the teeth were a peripheral dentinal layer, 30 microns thick with regular structure ("mantle dentine"), and a pronounced lamination of the rest of the dentine, which had an irregular structure with few tubules ("circumpulpal dentine").

The youngest tooth in the material—the upper second deciduous molar (+05), extracted when the patient was four years old—had abnormally thin dentine (Fig. 2). At the cervix the dentine was approximately 0.2 mm. thick, and below the fissure it was 0.13 mm. thick. The prenatal dentine consisted of two layers: the above-mentioned regular mantle dentine and an adjacent dark band with irregular structure (Fig. 3). Their combined thickness was

about 60 microns near one cusp. The postnatal dentine was lamellar with distinct dark lines separating lighter bands. In one cusp there were fifteen incremental layers with an average thickness of 32 microns.

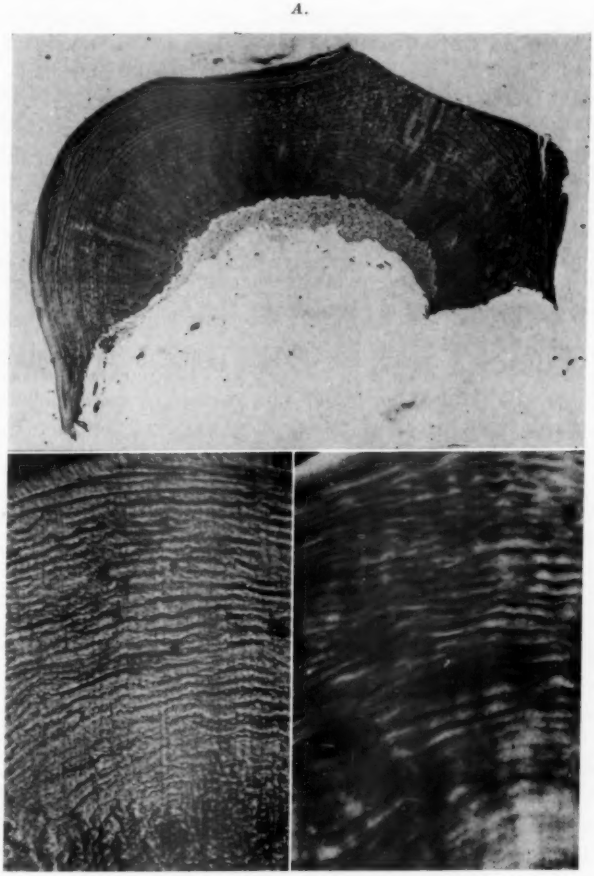


Fig. 4.—*A*, Decalcified section of the upper deciduous canine (03+) at 10 years. The dentine is relatively thick and exhibits abrasion and resorption. There are about forty-five lamellae. (Hematoxylin and eosin stain. Magnification, ×20; reduced ⅙.)

B, Unstained ground section of the lower first permanent molar (–6) in transmitted light. The tooth has fifty-six lamellae, with an average thickness of 33 microns. (Magnification, ×60; reduced ⅙.)

C, Microradiogram of the ground section shown in *B*. The alternating light and dark lines indicate rhythmic variations in the distribution of mineral salts in the dentine. (Magnification, ×60; reduced ⅙.)

The upper deciduous canines (+03 and 03+) were extracted when the patient was 10 years old. The dentine was similar in structure to that of tooth +05, but it was thicker (Fig. 4, *A*) and the number of incremental layers was found to be forty-three and forty-four, respectively, with an average thickness of 33 microns. The prenatal dentine was 150 microns thick in the tips of the

crowns. It consisted of regular "mantle dentine" and a dark layer of irregular dentine. The latter was approximately 120 microns thick and had no pronounced lamellar structure.

The lower second deciduous molar (–05), extracted when the patient was 11 years old, and the lower first permanent molar (–6), extracted at the age of 14 years, exhibited forty-five and fifty-six incremental layers in the cusps, respectively. The average thickness of the layers was 33 microns in both teeth. The incremental lamination was visible not only in decalcified sections but also in unstained ground sections (Fig. 4, *B*). Microradiographic examination showed that lamellae with high and low degrees of mineralization alternated in the dentine (Fig. 4, *C*).

DISCUSSION

By dividing the number of layers in the different teeth by the age of the patient at the time of extraction, practically the same result was obtained for all the teeth ($\frac{15}{4}, \frac{43}{10}, \frac{44}{10}, \frac{45}{11}, \frac{56}{14}$). If the lamellae in the dentine are taken as an expression of rhythmic formation, an average of four such layers would have been formed every year. If the rhythm was complete, the interval must have been three months.

As the incremental layers had an average thickness of 33 microns in the crowns, the daily rate of apposition was somewhat less than 0.4 microns. This is only one-tenth of the normal rate, which is said to be 4 microns in man.[11, 13] The slow rate of apposition resulted in a very thin dentine in the early life of the teeth. Thus, the upper second deciduous molar (+05) had a dentine thickness of only 0.2 mm. at the cervix at the age of 4 years, compared with the normal value of about 1.8 mm. The retardation of the growth of the dentine was compensated, however, by a prolonged development, as indicated by the radiographic and histologic examinations.

Attempts have been made to explain the peculiar structure of the dentine in osteogenesis imperfecta and in hereditary opalescent dentine in different ways. Such expressions as "dystrophy of the mesenchyme," "dysfunction of the odontoblasts," and "dedifferentiation" are often used. I do not intend here to discuss the mechanism, but it might be worth while to cite one of the latest contributions in this field. Ivancie[6] offers the following explanation of the lamellar structure of the dentine: The life span of the odontoblasts is severely curtailed, and after their degeneration are these short-lived odontoblasts replaced by other cells; this process of premature degeneration is repeated several times. According to Ivancie, the continued degeneration and replacement of odontoblasts can explain the unlimited production of dentine in hereditary opalescent dentine.

The calcification rhythm, which is normally 16 microns,[11] was strongly accentuated in the present material and was estimated to be 33 microns in three months. As it was impossible to exclude a priori the possibility that the calcification pattern of the dentine was a reflection of processes occurring in the

body that influenced the strength of the long bones, a study was made of the incidence of bone fracture. No connection could be demonstrated with certainty, however, between the mineralization rhythm and the fracturing of the bones.

SUMMARY

A boy with osteogenesis imperfecta had, up to the age of 13 years, suffered twenty-three bone fractures. The patient was under dental observation for thirteen years, beginning when he was 12 months old. Histologic examination of teeth extracted at various times suggested that the dentine had been formed rhythmically, probably at intervals of three months, so that a lamellar structure had arisen with an average layer thickness of 33 microns. The daily rate of dentine formation was estimated to be 0.4 microns, which led to a very thin dentine during the early life of the teeth. This retardation was partly compensated, however, by a prolonged development of the dentine. Microradiographic examination showed rhythmic variation in the degree of mineralization of the dentine, but no relationship could be traced between the mineralization rhythm and the fracturing of the bones.

In contrast to the deciduous teeth and the first permanent molars, the permanent incisors showed, on radiographic examination, obliterated pulp chambers already present at the age of 10 years. These incongruities are difficult to explain.

REFERENCES

1. Becks, H.: Histologic Study of Tooth Structure in Osteogenesis Imperfecta, Dental Cosmos **73**: 437, 1931.
2. Bergersen, B.: Om tanddannelse ved osteogenesis imperfecta, Norske Tannlaegeforen. Tid **39**: 503, 1929; **40**: 99, 1930.
3. Bergman, G.: Microradiography as a Method for Studying Dental Tissues, Odont. Revy **8**: 143, 1957.
4. Bergman, G., and Engfeldt, B.: Biophysical Studies on Teeth and Tooth-Germs in Osteogenesis Imperfecta, Acta path. et microbiol. scandinav. **35**: 537, 1954.
5. Engström, A., and Wegstedt, L.: Equipment for Microradiography With Soft X-rays, Acta radiol. **35**: 345, 1951.
6. Ivancie, G. P.: Dentinogenesis Imperfecta, Oral Surg., Oral Med. & Oral Path. **7**: 984, 1954.
7. Pindborg, J. J.: Dental Aspects of Osteogenesis Imperfecta, Acta path. et microbiol. scandinav. **24**: 47, 1947.
8. Rushton, M. A.: Structure of Teeth in Late Case of Osteogenesis Imperfecta, J. Path. & Bact. **48**: 591, 1939.
9. Rushton, M. A.: A New Form of Dentinal Dysplasia: Shell Teeth, Oral Surg., Oral Med. & Oral Path. **7**: 543, 1954.
10. Rushton, M. A.: Anomalies of Human Dentine, Ann. Roy. Coll. Surgeons England **16**: 94, 1955.
11. Schour, I., and Hoffman, M. M.: Studies in Tooth Development. I. The 16 Microns Calcification Rhythm in Enamel and Dentin From Fish to Man, J. D. Res. **18**: 91, 1939.
12. Schour, I., and Massler, M.: Studies in Tooth Development: The Growth Pattern of Human Teeth. Part II, J. Am. Dent. A. **27**: 1918, 1940.
13. Schour, I., and Poncher, H. G.: The Rate of Apposition of Human Enamel and Dentin as Measured by the Effects of Acute Fluorosis, Am. J. Dis. Child. **54**: 757, 1937.
14. Westin, G.: An Investigation Into Questions of Social Hygiene in the Counties of Västerbotten and Norrbotten, Sweden. Part III, chap. IV, Lund, 1937, Håkan Ohlssons Boktryckeri.
15. Winter, G. R., and Maiocco, P. D.: Osteogenesis Imperfecta and Odontogenesis Imperfecta, Oral Surg., Oral Med. & Oral Path. **2**: 782, 1949.

NEUROFIBROMATOSIS (VON RECKLINGHAUSEN'S DISEASE) WITH INVOLVEMENT OF THE MANDIBLE

Report of a Case

Stanley E. Winters, D.D.S., Horace F. Husser, M.D.,** Ernest E. Simard, M.D.,*** and Joseph J. Shebl, M.D.,**** Salinas, Calif.*

NEUROFIBROMATOSIS is a developmental disease, characterized by multiple tumors of the nerves. The purpose of this report is to show its effect upon the oral cavity and its associated structures.

CASE REPORT

A 32-year-old Mexican woman was referred for removal of impacted third molars and tumorlike growths in the mouth.

History.—The patient stated that she had had an enlargement of the undersurface of her tongue and the floor of her mouth for a number of years. This painless enlargement produced difficulty in mastication, for food would become caught between the enlargement and the lower teeth. Recently the tumor had not increased in size. The patient stated

Fig. 1.

Fig. 2.

Fig. 1.—The deformity at the angle of the right mandible and asymmetry of the face.
Fig. 2.—Note the polypoid lesions posterior to the lower anterior teeth.

From Salinas Valley Memorial Hospital, Salinas, California.
*Department of Oral Surgery.
**Chief of Staff.
***Department of Pathology.
****Department of Radiology.

that as a child she had undergone a resection of a portion of her right mandible, the exact nature of which she did not know. She thought it was following an attack of mumps.

Oral Examination.—Examination of this patient's mouth revealed a deformity at the angle of the right mandible (Fig. 1). The inside of the mouth revealed a villiform type of lesion occupying the entire undersurface of the tongue on the right side (Fig. 2). The lesion was quite soft, and the area felt velvety to palpation. Extending from the base of the undersurface of the tongue into the floor of the mouth was a similar growth. As the lesion approached the lower incisor area, where a bridge was located, there were two polypoid lesions measuring approximately 1.5 cm. in diameter. These polypoid lesions produced difficulty in mastication. The lesions were a little firmer than those on the undersurface of the tongue, but even they were quite soft and were covered by an intact mucous membrane.

General Physical Examination.—Numerous nodules were revealed in the intercutaneous and subcutaneous positions. These varied in consistency from soft to firm, and associated with them were coffee-colored spots of pigment over the extremities.

Laboratory Examination.—Laboratory findings were as follows:

Clotting time	4 minutes 20 seconds
Bleeding time	1 minute 20 seconds
Red blood cells	4,600,000
White blood cells	6,400
Nonsegmented	2
Segmented	69
Eosinophils	2
Basophils	2
Lymphocytes, total	24
Monocytes	1
V. D. R. L.	Negative
Urinalysis	Negative
Reaction	Acid
Specific gravity	1.010
White blood cells	1 to 3 per high-power field
Red blood cells	1 to 3 per high-power field
Epithelial cells	Some
Cast	0
Bacteria	Few
Crystals	0
Albumen	Negative
Sugar	Negative

Roentgenographic Examination.—The right mandible showed destruction of the inferior margin extending from the angle to the ramus anteriorly. The entire right mandible, including the symphysis of the mandible, showed irregular deformities, some with considerable absorption about them (Figs. 3, 4, 5, and 6).

The patient was seen by the Tumor Board of the Salinas Valley Memorial Hospital on July 12, 1956. It was the impression that this patient had Von Recklinghausen's disease or neurofibromatosis with involvement of the tongue and floor of the mouth. A biopsy was advised.

Operation.—On July 13, 1956, under general anesthesia, a biopsy specimen of the lesion was removed. An incision was made 4mm. distal to the lower right third molar area and, with the interproximal spaces serving as an incisal guide, extended to the right cuspid area. The mucous membrane was freed. This exposed an avascular dense, white, fibrotic type of tissue. A wedge of tissue approximately 2 cm. by 5 mm. was removed. The area of surgery was then closed with black silk sutures. The patient left the operating room in good condition.

Pathology Report.—

Gross description: The specimen consisted of two pieces of tissue, the largest of which was 2 cm. in greatest diameter. It was elliptical and had a smooth mucosal-covered surface. Projecting from this surface was a rounded nodule that was 1 cm. in maximum

Fig. 3.—Anteroposterior view showing the impacted teeth.

Fig. 4.—Destruction of right inferior margin of right mandible.

diameter and elevated a distance of 7 mm. On the cut surface this nodule was composed of uniform gray-white material that was slightly trabeculated. Two pieces of tissue were taken for microscopic study.

Microscopic study: The sections revealed the mass from the skin to be covered with a thin layer of epithelium, mildly keratotic and containing a considerable amount of melanin in the basement layer. The subepithelial area was quite thickened and presented interlacing groups of cells of small size. The lesion was moderately cellular. In some areas there was a mild suggestion of palisading, but for the most part this characteristic of a neurofibroma was not present. The lesion was poorly differentiated, and in no area did it present any unquestionable evidence of a neurofibroma, although it probably was such a lesion.

Fig. 5.

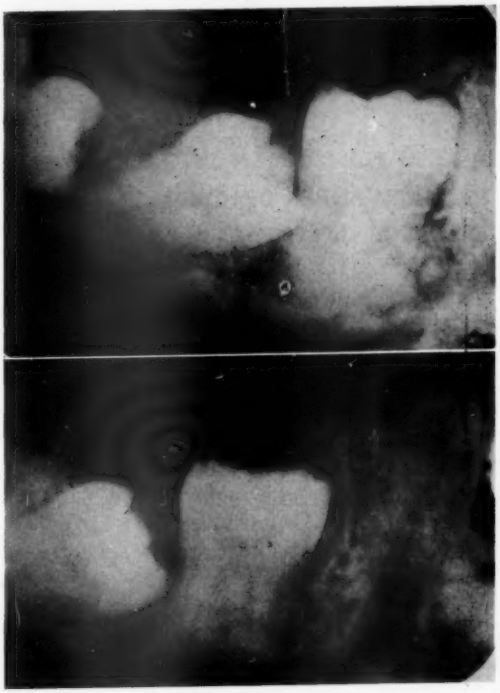


Fig. 6.

Fig. 5.—The position of the first, second, and third molars on the right side.
Fig. 6.—The position of the first and second molars on the left side.

The sections of the biopsies from the material projecting into the mouth revealed almost identical appearances. In no area was there any evidence of malignancy.

Diagnosis: Probably neurofibromatosis (Von Recklinghausen's disease).

CONCLUSION

It is concluded that this is a case of neurofibromatosis (Von Recklinghausen's disease). Neurofibromatosis may involve the mandible. Here, as elsewhere, the disease is not fatal and is best treated by removal of lesions that produce any difficulty, as in this case, with mastication.

ENDODONTICS

The American Association of Endodontists

Captain Warren J. Hedman, Editor

A BACTERIOLOGIC REPORT OF AN INVESTIGATION TO STUDY IN VIVO THE EFFECTIVENESS OF CERTAIN DRUGS FOR THE STERILIZATION OF CARIOUS DENTINE

Henry S. Schmidt, D.D.S., F.A.C.D., Mary C. Crowley, A.B., M.S.P.H., Verna Harner, B.S., and N. Weir Burkman, D.D.S., F.A.C.D., Ann Arbor, Mich.

THE effective sterilization of residual carious dentine, in deep-seated cavities, continues to be the objective of many dental research projects. The problem involved is to find an agent or a combination of agents which will effectively destroy or inactivate the microorganisms present in these lesions but not adversely affect the underlying pulpal tissue or the host. In 1954[1] we summarized results of an investigation on the use of a drug combination applied to carious dentine on a three-dentine-level treatment pattern. We continued this study, using only one application of the drug mixture after removing as much carious dentine as possible without making a mechanical exposure of the pulp. This was done to simulate clinical conditions as they confront an operator in his own office.

The study of the effect of sterilizing agents on deep seated caries in vivo presents many difficulties. Teeth that have deep carious lesions are usually so broken down that the maintenance of a sterile operative field is not easy. The procurement of desirable teeth for histologic sectioning after treatment presents still another problem. When teeth are comfortable, patients are reluctant to have them extracted. Human teeth present a wide variety of pulpal conditions and reactions because of age, previous injury by caries, trauma, and operative procedures. Thus, the effect of a medicament on the pulp presents a confusing picture which is difficult to evaluate correctly. This report is limited to the bacteriologic phase of the project described in the title.

This research project was conducted at the Kellogg Institute, University of Michigan Dental School, Ann Arbor, Michigan. The following constituted the research group: Drs. W. B. Eggleston, Sr., W. B. Eggleston, Jr., G. F. Weaver, Clarence Moyer, Wallace Niemann, William Bender, John Blakey, and Dale White. This group worked with the counsel of Drs. R. F. Sommer, F. D. Ostrander, and D. A. Kerr of the University of Michigan Dental School.

Because of the undeterminable location[2-6] of microorganisms in carious lesions, the use of an effective germicidal agent is desirable in cavities and particularly in cavities that approximate the pulp.

NONSPECIFIC THERAPY

The value of phenol as a sterilizing agent for carious dentine has been debated for years. When phenol is placed in a cavity, its primary action is the coagulation of the protein contents of the dental tubules. The main disagreement regarding phenol's action is over whether or not the drug is self-limiting, that is, whether or not it forms an impenetrable coagulum at the orifice of the dental tubule. While phenol might possibly destroy bacteria that it can reach in sufficient concentration, there is still considerable doubt as to its ability to penetrate dentine for any distance.[7-9] When phenol is used in deep cavities, there is the added possibility of its doing damage to the underlying pulpal tissues. What is true for phenol is also true for phenol derivatives in varying degrees.

The use of zinc oxide-eugenol has been advocated in recent years for the sterilization of deep-seated residual caries.[10] Miller[11] first discredited eugenol as a sterilizing agent. More recently Turkheim[12] reported that zinc oxide-eugenol in no single instance sterilized disks of naturally carious dentine. James and Schour[13] reported in a study on human teeth that "under cavities filled with zinc oxide and eugenol, the pulp showed only minimal or occasional inflammation. There was concurrently an absence of reparative dentin." The sedating of the pulp in teeth with deep-seated residual caries, without destroying or inactivating the microorganisms present, is of no therapeutic value. The formation of reparative dentine is not only valuable to the strength of the remaining tooth structure but also indicates the return to a normal physiologic function of the pulpal tissues involved.

Silver nitrate, an escharotic similar to phenol, coagulates the protein contents of the dental tubule. Its ability to penetrate carious dentine in sufficient concentration to be effective as a sterilizing agent[8, 14] is limited. Perreault and associates,[15] in an in vivo study on the rat incisor, found that silver nitrate penetrated normal dentine rapidly and caused damage to the pulp. Others[16, 17] have found that silver nitrate does penetrate sound human dentine and the products of its precipitation eventually react on the pulp. When silver nitrate is applied to deep residual caries in sufficient concentration, and for the length of time necessary to be effective, there is a probability of its doing damage to the pulpal tissue.

CHEMOTHERAPEUTICS

Ideally, antibiotic therapy is the treatment of infection by administration of certain cellular products and/or chemicals which affect the causative organisms unfavorably but do not injure living tissue. For dentine sterilization

this form of therapy includes those drugs which adversely affect the microorganisms but do not adversely affect the contents of the dental tubules or the pulp.

Camphorated parachlorophenol and soluble nonbuffered penicillin G were used in this study for the following reasons:

1. They possess a wide bactericidal spectrum. Penicillin is effective against most Gram-positive and some Gram-negative organisms. Camphorated parachlorophenol is a nonspecific drug which is effective against a wide variety of microorganisms.

2. Penicillin is neutralized by penicillinase. The latter can be added to culture media so that reliable cultures may be obtained. Camphorated parachlorophenol is not inhibitory after forty-eight hours' exposure of organisms to the drug.

3. Penicillin and camphorated parachlorophenol are tolerated by the affected tissues.

The drug mixture is made by placing a 50,000 unit tablet of soluble penicillin G (nonbuffered) on a glass slab and mixing the penicillin with enough camphorated parachlorophenol to make a dry, stiff paste.

EXPERIMENTAL PROCEDURE

When this investigation was started, each case was studied on an ingressive three-treatment basis. This was done in an attempt to evaluate the penetrability and effect of the drugs on carious dentine. At the first appointment, a culture was taken to establish the presence of microorganisms. Then approximately the outer third of the carious dentine was removed and the drug mixture was sealed in. At the second appointment, the middle layer of carious dentine was first cultured and then removed. Following this, the second drug treatment was applied and sealed in. At the third appointment, the deepest layer of carious dentine was cultured and removed. Care was taken not to expose the pulp. Then the third treatment was applied and sealed in. These treatments were approximately one week apart.

In the latter part of the experiment an attempt was made to associate this technique with procedures that could be used in everyday practice. As much carious dentine as possible was removed without creating a pulpal exposure, following which the drug mixture was applied to the remaining carious tooth structure.

DISCUSSION

When the foregoing technique is employed, the first consideration should be the health of the patient and the condition of the tooth or teeth to be treated. All subjective symptoms, as well as clinical evidence, should be carefully and thoroughly considered in an attempt to evaluate properly the existing condition of the pulp involved. Specifically, pulpal healing is directly proportional to the

amount and quality of the blood available. Other factors to be considered are occlusal trauma, previous operative procedures, and the presence of inflammatory symptoms or conditions.

When the drug combination is applied to carious lesions near the pulp, the patient usually experiences a mild pain sensation for about five minutes. This pain is associated with the liquefaction of the drug after its application. If neither pain nor liquefaction of the drug occurs, the pulp probably contains either a poor circulation or none at all and consequently requires a different type of treatment.

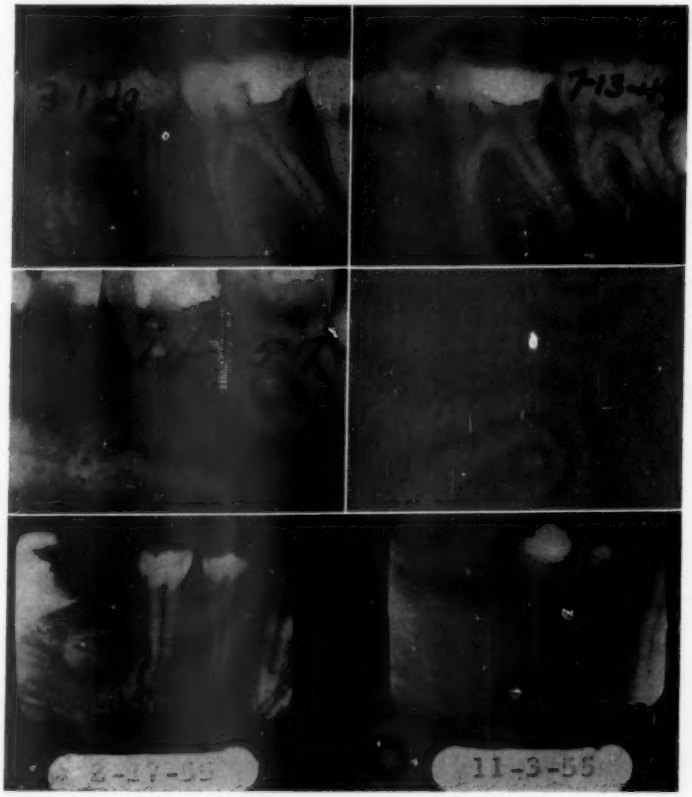


Fig. 1.—Teeth showing periapical areas returning to normal following treatment.

Whether the molecules of penicillin and camphorated parachlorophenol advance toward the pulp by means of osmotic action, simple diffusion, or lymphatic circulation has not been determined. The fact that these drugs can and do penetrate dentine has been demonstrated by both Shuttleworth[18] and Stamps.[19]

SEALING-IN OF THE DRUGS

The method of sealing-in of these drugs in carious dentine by the use of an inert material is entirely new. This permits a reservoir of the drug mixture to be held against the carious dentine for as long as it is effective.

The following materials for sealing in the drugs are suggested:

1. A mixture of heavy magnesium oxide powder, 50 per cent, and powdered white rosin, 50 per cent, with sufficient chloroform to produce a tacky mix.

2. A mixture of heavy magnesium oxide, 25 per cent, powdered white rosin, 25 per cent, and Wonderpak powder, 50 per cent, plus sufficient chloroform to make a thick, sticky paste.

3. A thin layer of gutta-percha or wax.

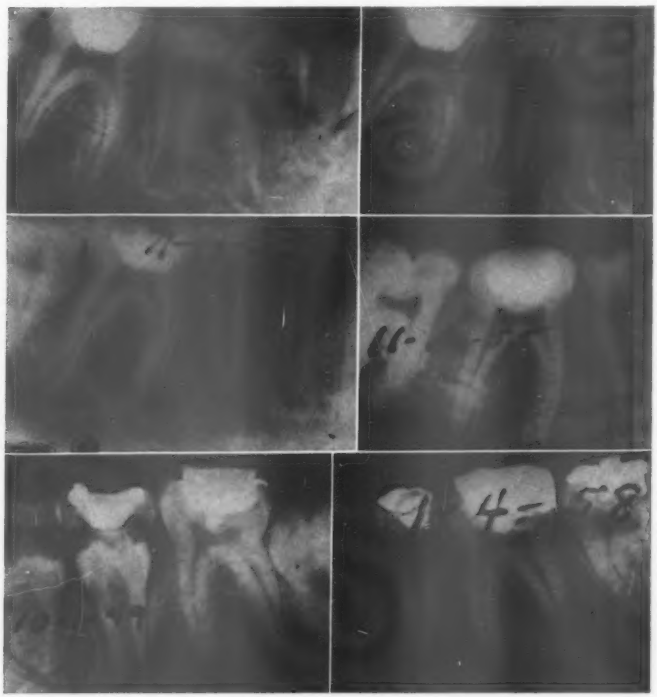


Fig. 2.—Teeth showing limited formation of reparative dentine following treatment.

In all these methods the cavity margins should be wiped dry with sterile cotton pellets or cotton moistened with chloroform, so that the sealer may be tacked down on dry, noncarious tooth structures.

After a thin layer of the sealer has been secured over the dentine to be sterilized, a layer of soft cement is applied, avoiding pressure on the pulp.

Following this, the tooth may be restored to physiologic function by a method of the operator's choice.

The first and second methods are the methods of choice. The third was the original one used and should be considered only an alternative method.

Clinically, the following changes were observed after treatment:

1. The dentine became dehydrated and firm.
2. Vitalometric tests were generally improved.
3. Roentgenographically, many teeth showed periapical areas indicative of early inflammatory changes in the pulp returned to normal.[1]
4. There was a limited formation of reparative dentine.

BACTERIOLOGY

The bacteriologic methods were the same as those previously described.[1] All negative cultures were incubated for one week at 37.5° C. before being discarded.

The bacteriologic results over a six-year period are summarized in Table I. A total of 146 teeth were treated. Of these, 87 per cent were sterile after one to three treatments.

TABLE I. SUMMARY OF BACTERIOLOGIC RESULTS FOR SIX YEARS

YEAR	NUMBER OF TEETH TREATED	NUMBER NEGATIVE AFTER ONE TO THREE TREATMENTS	NUMBER POSITIVE AFTER THREE TREATMENTS	PER CENT NEGATIVE AFTER THREE TREATMENTS
1952-53	44	33	11	75
1953-54	20	18	2	90
1954-55	31	27	4	87
1955-56	18	16	2	88
1956-57	12	12	0	100
1957-58	21	21	0	100
Total	146	127	19	87

Table II shows the bacteriologic results after removal of as much carious dentine as possible in one operation. Thirty-three teeth were treated. Of these, 79 per cent were negative after one treatment, and none was positive after three treatments.

TABLE II. BACTERIOLOGIC RESULTS AFTER REMOVAL OF AS MUCH CARIOUS DENTINE AS POSSIBLE IN ONE OPERATION

NUMBER OF TEETH TREATED	NUMBER NEGATIVE AFTER ONE TREATMENT	NUMBER NEGATIVE AFTER TWO TREATMENTS	NUMBER NEGATIVE AFTER THREE TREATMENTS
33	26 (79%)	6 (18%)	1 (3%)

Table III presents a summary of the controls used and bears out the findings of Besic[20] and others that changing the environment will destroy cariogenic bacteria in certain cases.

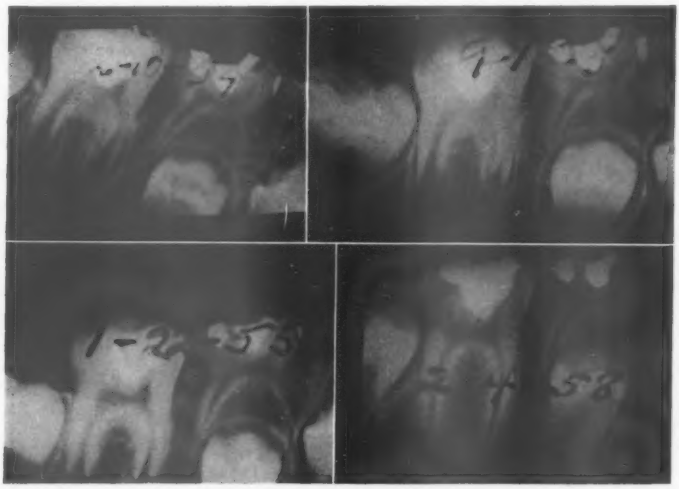
TABLE III. CONTROLS (NO DRUG USED)

SEALER	NUMBER OF TEETH TREATED	NUMBER NEGATIVE AFTER ONE TREATMENT	NUMBER POSITIVE AFTER ONE TREATMENT
Cement only	7	2	5
Magnesium oxide, rosin, chloroform, and cement	5	3	2
Gutta-percha and cement	3	0	3
Total	15	5 (33⅓%)	10 (66⅔%)

SUMMARY

A method has been described by which selected teeth with deep carious lesions may be restored to physiologic function. If this drug combination can be applied to teeth in which only early inflammatory reactions are present in the pulp, a large percentage of these teeth may be saved and restored to physiologic function.

A.



B. C.

Fig. 3.—Extreme case demonstrating pulpal repair following treatment for carious dentine (*A*), return to normal physiologic function as demonstrated by continued root formation (*B*), and the value of a good blood supply (*C*).

The extent of the pulpal breakdown, the condition of the circulation, and the virulence of the invading bacteria must determine the point from which there is no possible return to normal.

CONCLUSIONS

1. Eighty-seven per cent of the teeth cultured throughout the experiment did not show growth after three treatments.

2. In the latter part of the experiment 79 per cent of the teeth cultured did not show growth after one treatment.

3. This form of chemotherapy offers a promising approach to cavity sterilization.

REFERENCES

1. Burkman, N. W., Schmidt, H. S., and Crowley, M. C.: A Preliminary Report of an Investigation to Study the Effectiveness of Certain Drugs for Sterilization of Carious Dentine, ORAL SURG., ORAL MED. & ORAL PATH. **7**: 647, 1954.
2. Dorfman, A., Stephan, R. M., and Muntz, J. A.: In Vitro Studies on Sterilization of Carious Dentin. II. Extent of Infection in Carious Lesions, J. Am. Dent. A. **30**: 1901, 1943.
3. Kronfeld, R.: Histopathology of the Teeth and Their Surrounding Structures, ed. 3, Philadelphia, 1949, Lea & Febiger, p. 144.
4. Miller, W. D.: Micro-Organisms of the Human Mouth, Philadelphia, 1890, S. S. White Dental Mfg. Co., pp. 179-180.
5. Seltzer, S.: The Bacteriologic Status of Dentin After Cavity Preparation, J. Am. Dent. A. **27**: 1799, 1940.
6. Zander, H. A.: Bacteria in Dentin After Cavity Preparation, Illinois D. J. **9**: 207, 1940; Der Zahnartz, J. **1**: 375, 1940.
7. Seltzer, S.: The Comparative Value of Various Medicaments in Cavity Sterilization, J. Am. Dent. A. **28**: 1844, 1941.
8. Stephan, R. M., Muntz, J. A., and Dorfman, A.: In Vitro Studies on Sterilization of Carious Dentin, III. Effective Penetration of Germicides Into Carious Lesions, J. Am. Dent. A. **30**: 1905, 1943.
9. Thomas, B. O. A.: Penetration of Phenol in Tooth Structures, J. D. Res. **20**: 435, 1941.
10. Massler, M.: Sterilization of Dentin, J. Tennessee D. A. **35**: 375, 1955.
11. Miller, W. D.: On the Comparative Rapidity With Which Different Antiseptics Penetrate Decalcified Dentin, Dental Cosmos **33**: 337, 1891.
12. Turkheim, H. J.: In Vitro Experiments on the Bactericidal Effect of Zinc Oxide Eugenol on Bacteria-Containing Dentin, J. D. Res. **34**: 295, 1955.
13. James, V. E., and Schour, I.: Prepublished Abstracts, J. D. Res. **34**: 25, 1955.
14. Hardwick, J. L.: Sterilization of Carious Dentin, Proc. Roy. Soc. Med. **42**: 815, 1949.
15. Perreault, J. G., Massler, M., and Schour, I.: Reaction of Odontoblasts to Medicaments Placed in Cavity Preparations in Rat Incisors, J. Am. Dent. A. **52**: 533, 1956.
16. Zander, H. A., and Burrill, D. Y.: Penetration of Silver Nitrate Solution in Dentin, J. D. Res. **22**: 85, 1943.
17. Englander, H. R., James, Verda E., and Massler, M.: Histologic Effects of Silver Nitrate on Human Dentin and Pulp, J. Am. Dent. A. **57**: 621, 1958.
18. Shuttleworth, C. W.: The Diffusion of Penicillin From the Dental Root Canal, Brit. D. J. **89**: 127, 1950.
19. Stamps, H. F.: The Testing of Dentin Sterilizing Agents, Univ. Michigan D. School Alumni Bull. **55**: 16, 1953.
20. Besic, F. C.: The Fate of Bacteria Sealed in Dental Cavities, J. D. Res. **22**: 349, 1943.

DISCUSSION (HERBERT SCHILDER, D.D.S., B.A., BOSTON, MASS.)

I would like to thank the authors for having presented a very provocative paper which raises many important points regarding preventive endodontics. Preventive endodontics is a subject in which Dr. Burkman and his associates are particularly interested and one subject which we, too, should all view with enthusiasm.

It is for this reason that we are always happy to hear of the latest findings of this group concerning their very attractive idea. The idea, as I understand it, is to treat teeth with deep carious lesions—carious lesions that are deep enough to involve the pulp—without exposing the pulp and somehow to halt the carious process and repair the involved pulp chemotherapeutically.

From a public health point of view, the idea has extraordinary appeal.

Historically, this goal has been sought for many years, and many drugs have been used to achieve sterilization of dentine walls in deep cavities. As

the present authors have pointed out, this sterilization should be harmless to the pulp and ideally it should be therapeutic.

The current paper certainly seems to bear out earlier reports by the group that a combination of penicillin and camphorated parachlorophenol does achieve a high degree of sterilization of deep layers of infected dentine. Other questions arise, however, which must be answered experimentally before complete acceptance of this technique can be achieved.

The authors are aware, of course, of many of these other questions, and I know that they plan to pursue several of them in the near future.

It might be well to point out here some of the complicating factors involved in this type of work.

First, in a study of this type, we should use only teeth in which the decay has actually involved the pulp. This cannot be determined conclusively with a radiograph. If sound dentine remains between the floor of the cavity and the pulp, the percentage of apparently successful cases will be increased appreciably. If sound dentine does exist, the simplest and best treatment would be complete excavation. If, on the other hand, the caries actually involves the vital pulp, this can be determined only by excavation to the point of exposure, thus causing the tooth to be useless for the study. Tooth selection for significant results is admittedly very difficult but also very important.

Second, as Schmidt and Burkman pointed out in *Dental Clinics of North America,** does surface sterilization necessarily mean sterilization in *depth?* And, as they have pointed out previously,** does drug penetration necessarily mean effective *depth* of sterilization?

Third, from the point of view of pathology, what is the significance of the initial five-minute painful response?

Fourth, if the penicillin and camphorated parachlorophenol actually work a therapeutic change on the involved pulp, what are the future consequences of this reaction? Will accelerated aging processes (pulp stones and internal resorption) occur as they are often seen to do with the use of calcium hydroxide?

Fifth, assuming that the technique is a successful one, exactly how limited is its application with regard to the health of the patient, the age of the tooth, and the amount of closure of the root apex?

Finally, everyone familiar with this important work done by the group at the University of Michigan continues to await the complete pulpal histopathologic evidence to support this procedure. We eagerly look forward to the time when their work can finally be substantiated by controlled microscopic study of sections of treated teeth.

Negative cultures are one thing; histologic evidence may be of even greater significance. I know that Dr. Burkman and his associates are as anxious to see sections of these teeth as we are, and I hope that they will soon have them available for study.

Until that time, I would like to thank them again for an excellent presentation of the bacteriologic aspects of this problem and to wish them success with this very worth-while project.

---

*Burkman, N. W., and Schmidt, H. S.: Cavity Medication: A Review, D. Clin. North America, pp. 165-177, March, 1958.

**Burkman, N. W., Schmidt, H. S., and Crowley, M. C.: A Preliminary Report of an Investigation to Study the Effectiveness of Certain Drugs for Sterilization of Carious Dentine, ORAL SURG., ORAL MED. & ORAL PATH. 7: 647, 1954.

RESEARCH

. . . . . .

HEALING OF GINGIVAL MUCOPERIOSTEAL FLAPS

Charles A. Kohler, D.D.S., M.S., and Sigurd P. Ramfjord, L.D.S., M.S., Ph.D., Ann Arbor, Mich.

INCISION and elevation of the gingival mucoperiosteum is used routinely in conjunction with extractions, periodontal flap operations, and other surgical interventions on the alveolar process.[2] It is generally assumed that such surgical flaps will heal completely following proper repositioning, but this contention is based on rather limited scientific evidence. Workman,[15] in two patients, and Irwin,[5] in four patients, found histologic evidence of complete healing following surgical deflection and repositioning of mucoperiosteal flaps. Complete healing, without microscopic documentation, in two human subjects also was reported by Borden.[1] A comprehensive study of healing following surgical detachment of human periodontal tissues from the teeth was reported a few years ago by Morris,[8] although the narrow surgical pockets which he created were not entirely comparable to gingival mucoperiosteal flaps. Several studies of healing following flap operations also have been undertaken in animals.[1, 3, 12, 13] It has been reported that in both human beings and animals postoperative healing depends upon (1) the extent of curettage and injury to the exposed root surfaces, (2) the vitality of the teeth if dentine has been exposed,[9] (3) the degree of presurgical inflammation associated with the periodontal pockets, and (4) the protection of the areas of surgery.

Fish,[3] for example, found no healing of pockets which were created surgically in areas of gingivitis in rhesus monkeys. Stones[13] and Skillen and Lundquist[12] found partial healing at the bottom of such pockets in monkeys and dogs. Glickman and Lazansky,[4] using dogs, and Ramfjord,[10] using monkeys, reported complete healing following experimental separation of periodontal structures from the root surfaces. Various degrees of healing following a variety of surgical flap procedures and pocket extensions also were reported by Linghorne and O'Connell[6-7] and Waerhaug.[14] The consensus reflected in the literature is that complete healing has been obtained when the surfaces of the root have not been curetted or injured. In most cases some attachment has been lost when the root surfaces have been curetted.

From the University of Michigan, School of Dentistry.

The purpose of this study was to investigate, clinically and histologically, the healing of surgical mucoperiosteal gingival flaps in human beings, avoiding any deliberate injury or curettage of the root surfaces.

MATERIAL

The experimental material was obtained from patients who needed complete extraction of the remaining teeth in at least one of the dental arches. The subjects were clinically healthy patients ranging in age from 16 to 68 years. A thorough examination of the periodontium was performed on each subject. An attempt was made to include in the experiment areas of clinically healthy gingival tissues as well as areas exhibiting various degrees of gingival inflammation. Areas with advanced periodontal destruction were not included, since it would be difficult to obtain satisfactory specimens for histologic investigation from such areas. Fifteen specimens were obtained from fourteen patients. Fourteen of these fifteen specimens were obtained from the maxillary anterior region because of the ease of access and the possibility of minimizing the surgical defect in this region. Roentgenograms and intraoral photographs of all patients were available.

METHOD

A horizontal notch was made with a diamond stone on the labial surface of the experimental tooth, 1 to 3 mm. incisally to the gingival margin. With cotton pliers, a Boley gauge, and the sharp end of No. 2 silver point of the type used for root canal therapy, measurements were recorded from the gingival border of the notch to the free gingival margin and from the notch to the bottom of the gingival crevice or pocket. At the time of extraction of the teeth adjacent to the experimental tooth, an incision was made in the interdental papillae at each side of the tooth to be studied, and a labial mucoperiosteal flap was raised and reflected away from the tooth and alveolar process. An attempt was made to duplicate the routine procedures used in oral surgery and periodontics for doing a gingival mucoperiosteal flap. The distance from the notch to the alveolar crest was recorded. A small gingival biopsy specimen was secured from an area where the clinical condition closely resembled the experimental tooth. The purpose of this biopsy was to study histologically the gingival tissues of the patient at the time of the flap operation. The mucoperiosteal gingival flap was sutured back into its previous position and the healing was observed clinically at various intervals of time until the patient was ready for extraction of the experimental tooth.

Before the experimental tooth was removed, a clinical examination was performed, duplicating the procedures of the examination undertaken prior to the flap procedure. New intraoral photographs were obtained, and the measurements from the notch to the free gingival margin and from the notch to the bottom of the gingival sulcus were recorded as posthealing measurements.

Then block sections were removed. These sections included a part of (1) the labial gingiva, (2) the underlying alveolar process, (3) the periodontal

membrane, and (4) the tooth. Block sections of this type have to be removed very carefully with sharp instruments to prevent separation of the gingival tissues from the tooth and the alveolar process. Fig. 1 illustrates the procedure for obtaining such block sections. Incisions are made through the periosteum to the bone from points *A* to *C*, *B* to *D*, and *C* to *D*. The vertical incisions should be carried 5 to 10 mm. apically to the alveolar crest. The larger the area that is included in the specimen the less tendency there is for separation of the tissues. Starting on the crest of the alveolar ridge at point *B*, another incision is made which sweeps posteriorly and superiorly to the mucobuccal fold at point *E*. A mesially directed vertical gingival incision is made from *A* to *F*, and the two flaps (*C*, *A*, *F*, and *D*, *B*, *E*) are deflected in the direction of the arrows until they are completely separated from the specimen represented by *A*, *C*, *D*, *B*. The alveolar process is cut with a tapered fissure bur parallel to the lines of the original mucoperiosteal incision *A-C*, *B-D*, and *C-D*, and about 1 to 2 mm. away from these lines. These cuts must be made into the tooth to assure the intact removal of the specimen, and the cut from points *C* to *D* is extended through the root of the tooth to separate the specimen from the apical part of the root. An elevator is inserted into the horizontal groove through the root, and the coronal part of the tooth, with the alveolar process and the gingival tissue, is removed by rotation of the elevator. No forceps are used.

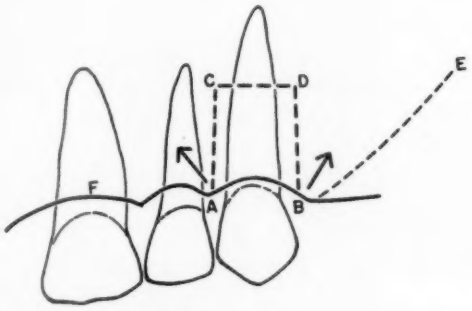


Fig. 1.

It is very important that due consideration be given to prevention of post-surgical defects in the area from which the specimen is removed. The distal flap represented in Fig. 1 by *E*, *B*, *D*, must be reflected beyond the attached gingiva until loose connective tissue can be felt between the periosteum and the alveolar mucosa. Then the periosteum is severed with a sharp knife across the entire width of the base of the flap. This cut should extend barely through the periosteum. The elasticity of the mucous membrane and the underlying soft tissues will allow the flap to be pulled anteriorly. The mesial flap represented by *C*, *A*, *F*, in Fig. 1 also is loosened from the alveolar process over a considerable area. The margins of the two flaps are then approximated and sutured together, and the defect is covered both by the mucous membrane and the periosteum of the adjacent flaps.

The patients were seen for final control about one year after the specimens had been removed. No residual defects could be observed. From clinical examination, it was impossible to tell from which area the specimen had been removed. Fig. 2, *A*, *B*, and *C* illustrate the clinical findings of a typical case before and after surgery. Fig. 2, *D* is added to show another typical post-operative area.

Fig. 2.—*A*, Case 4 prior to flap operation. *B*, Case 4 prior to removal of specimen. Complete healing following flap operation. *C*, Case 4 eleven months after removal of the specimen from the maxillary right cuspid area. No residual defect. *D*, Case 6 thirteen months following removal of a block specimen in the region of the right maxillary cuspid. No residual defect.

Probably one reason that so few investigations of this type have been undertaken in human subjects is the fear of residual defects. When the described technique was used, satifactory specimens were obtained for this study without any harm to the patients. A number of initial specimens had to be discarded because of artifacts associated with attempts to remove too small a specimen until the reported technique was worked out in detail.

The specimens were fixed in formalin, decalcified, and imbedded in celloidin. A large number of sections for microscopic study were prepared from each block. The sections were stained with hematoxylin and eosin and with azan stains for connective tissues.

CLINICAL OBSERVATIONS AND CLINICAL AND HISTOLOGIC MEASUREMENTS

Satisfactory clinical healing followed the flap operation in all the cases investigated. Clinically, the healing appeared to be by first intention in fourteen of the fifteen cases. In one patient a thin, nonpurulent discharge could be expressed from beneath the flap when the sutures were removed three days after the flap procedure. There was no clinical evidence of healing or reattachment of the flap to the alveolar process or the tooth at this time, but complete clinical healing occurred later. No difference in rate or degree of healing could be observed between areas of gingivitis and areas of normal gingiva. The rate or degree of healing was the same for teeth in heavy occlusion, teeth in normal occlusion, and teeth with no antagonists.

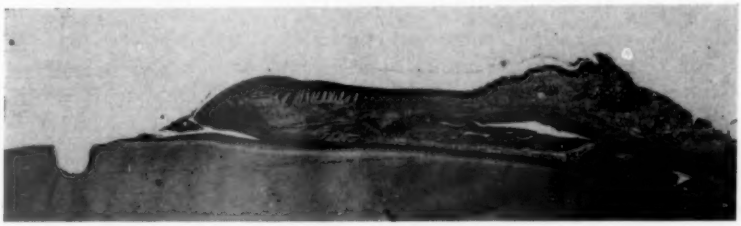


Fig. 3.—Case 13. Section from typical specimen. The notch extends into the dentine. (Magnification, ×7.)

The clinical and microscopic measurements have been compiled in Table I. The measurements listed in the "presurgical" columns were obtained immediately prior to the flap operation. A No. 2 very thin silver root canal point was used to probe the depth of the gingival crevice and, unfortunately, in some cases anesthesia had been administered prior to the probing. The measurements from notch to alveolar crest were obtained during the flap operation after the alveolar crest had been exposed by deflection of the mucoperiosteal flap. The figures recorded in the first two "posthealing" columns were obtained immediately before the surgical removal of the block section.

The data in the third and fourth "posthealing" columns are from the prepared tissue sections (Fig. 3). A calibrated eyepiece for the microscope

was used, and the average of three measurements obtained from different sections of each specimen was recorded. In some cases microscopic measurements could not be obtained, and corresponding vacancies exist in these columns. The vacancies exist because the notch made initially in the tooth did not enter the dentine, and this landmark was lost with the enamel during the process of decalcification.

A comparison between the various measurements can be found in the four columns under "results." The figures in the first of these columns are obtained by comparison of the figures in the first "presurgical" column and the figures in the first "posthealing" column. If the posthealing measurements are greater than the presurgical measurements, a loss in height of the free gingival margin has occurred. This loss is indicated by a minus sign (–) preceding the number representing the loss in millimeters. Plus signs (+) are used in a similar manner to indicate gain in height of the gingival margin. The "depth of sulcus" results are obtained by a comparison of the figures in the second "presurgical" column and the figures in the second "posthealing" column. An increase in depth of the gingival sulcus relative to the notch is recorded by a minus sign in front of the numerical values, which are expressed in millimeters. Conversely, a plus sign is used to indicate a more coronal position of the bottom of the sulcus following the healing than prior to the flap procedure.

In order to check the validity of the clinical measurements, a comparison was made between the clinical measurements in the second "posthealing" column and the microscopic measurements in the third "posthealing" column. The results are listed in the third "results" column. When the clinical measurements exceeded the microscopic measurements, the discrepancy in millimeters is listed with a plus sign; when the clinical measurements are less than the microscopic measurements, a minus sign is used to indicate the discrepancy. When the two measurements are the same, a zero designation is used. For the cases for which data are available the distances from the notch to the alveolar crest measured clinically during the flap operation are compared with the distances from the notch to the alveolar crest measured microscopically in the posthealing specimens. The difference in measurements is listed in the last column of the table, under "alevolar crest." A plus sign indicates that the distance measured appeared greater clinically than microscopically, and a minus sign is used to designate a shorter clinical than microscopic measurement. These measurements, of course, are not directly comparable because of the variation in time.

The variations of the position of the free gingival margin observed before and after the flap procedure are within the limits reported by Morris[8] and also by Irwin.[5] In four instances the free gingival margin was found to be from 0.2 to 0.8 mm. closer to the notch than at the time of the flap operation. In nine instances the free gingival margin was found to be from 0.1 to 1.2 mm. more apically positioned than in the initial presurgical measurements. In two instances no change was observed in the position of the free gingival margin. The measurements for depth of the gingival crevice showed a slight increase in six cases, from 0.2 to 1.2 mm., and a decrease in depth in seven cases, ranging from

0.1 to 1.2 mm.; in two cases no change of depth was observed. In thirteen of the fifteen cases the measured change in depth of the gingival sulcus was less than 1 mm.

The correlation between the clinical and microscopic measurements of the depth of the sulcus is of some interest. If it is assumed that the probe reached the connective tissue at the bottom of the epithelial attachment, one would expect the clinical measurements to be slightly greater than the microscopic measurements since the clinical measurements were made from the enamel border of the notch and along the surface of the enamel to a point on the root surface and since the microscopic measurements were made from the dentinal border of the notch to the same point on the root surface. Assuming that the walls of the notch are perpendicular to an extension of the root surface, the hypotenuse of a triangle extending to the enamel surface of the notch would be longer than the hypotenuse of a triangle starting on the surface of the dentine (the point on the root being the same in both instances). Two obvious errors in the clinical measurements resulted from the use of the very thin probe in the gingival crevices that had been anesthetized before the probing was done. In Cases 1 and 6 (Table I) the silver probe obviously had been pushed down to the alveolar crest, penetrating the soft tissues. This explains the discrepancies of 1.2 and 1.8 mm. between the microscopic and the clinical measurements. In the rest of the cases a remarkably good correlation was found between the microscopic and the clinical measurements, with variations from 0 to 0.4 mm. The obvious lessons to be learned from this are that a sharp-pointed probe should not be used for measuring pocket depth and that the pocket depth should always be measured before anesthesia is induced.

The clinical measurements of the distance between the notch and the alveolar crest at the time of the flap procedure equaled fairly well the microscopically measured distance between the notch and the alveolar crest in the posthealing specimens. The greatest decrease in height of the alveolar crest (0.8 mm.) was noted in Case 15. Microscopically, the slides from this specimen also showed a marked active resorption at the alveolar crest.

Considering the discussed errors in our methods of measuring, it can be stated that no signficant difference was found between the position of the free gingival margin, the bottom of the gingival sulcus, and the position of the alveolar crest before and after flap procedures.

HISTOLOGIC FINDINGS

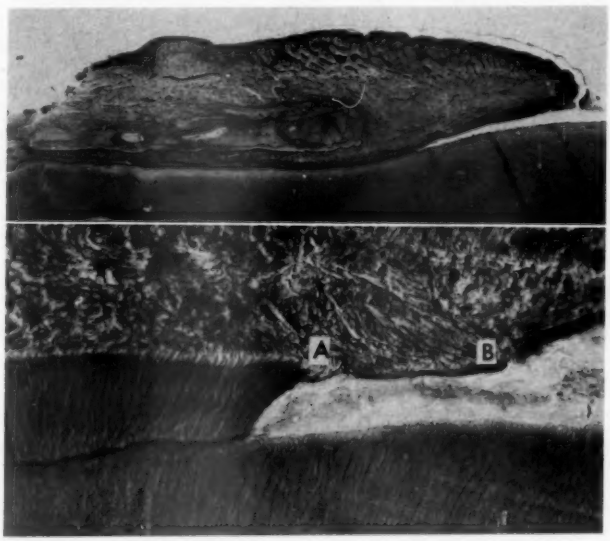
Microscopic examination of the specimens confirmed the clinical observation that healing followed the described flap procedure in all of the fifteen cases examined. A complete union of the surgically separated structures was observed, and the functional arrangement of the gingival and alveolar crest fibers was re-established. In Case 13 the flap was not attached to the tooth and alveolar process at the time of the removal of the sutures, but later clinical and histologic examinations revealed that complete healing had occurred in this case.

TABLE I

CASE	AGE OF PATIENT (YEARS)	TOOTH	PRESURGICAL: NOTCH TO FREE GINGIVAL MARGIN (MM.)	PRESURGICAL: NOTCH TO BOTTOM OF GINGIVAL SULCUS (MM.)	NOTCH TO ALVEOLAR CREST (MM.)	HEALING INTERVAL (DAYS)	POSTHEALING: NOTCH TO FREE GINGIVAL MARGIN CLINICAL (MM.)	POSTHEALING: NOTCH TO BOTTOM GINGIVAL SULCUS CLINICAL (MM.)	POSTHEALING: NOTCH TO BOTTOM EPITHELIAL ATTACHMENT MICROSCOPIC (MM.)	POSTHEALING: NOTCH TO ALVEOLAR CREST MICROSCOPIC (MM.)	RESULTS GAIN (+) OR LOSS (−): GINGIVAL MARGIN CLINICAL (MM.)	RESULTS GAIN (+) OR LOSS (−): DEPTH OF SULCUS CLINICAL (MM.)	RESULTS GAIN (+) OR LOSS (−): CORRELATION OF CLINICAL AND MICROSCOPIC MEASUREMENTS: DEPTH OF SULCUS (MM.)	RESULTS GAIN (+) OR LOSS (−): CORRELATION OF CLINICAL AND MICROSCOPIC MEASUREMENTS: ALVEOLAR CREST (MM.)
1	18	$\overline{3\vert}$	3.0	4.0	—	14	3.0	3.9	2.7	4.5	0.0	+0.1	+1.2	—
2	16	$\underline{1\vert}$	1.5	4.2	6.1	28	2.4	5.1	—	—	−0.9	−0.9	—	—
3	34	$\vert\underline{3}$	1.0	4.6	4.6	28	1.7	3.9	3.5	4.7	−0.7	+0.7	+0.4	−0.1
4	27	$\underline{3\vert}$	1.2	3.5	3.7	85	0.4	3.7	—	—	+0.8	−0.2	—	—
5	58	$\vert\underline{3}$	2.6	4.0	4.4	88	1.8	4.0	4.0	5.0	+0.8	0.0	0.0	−0.6
6	48	$\underline{3\vert}$	1.0	3.2	4.4	94	1.1	4.4	2.6	4.0	−0.1	−1.2	+1.8	+0.4
7	27	$\underline{3\vert}$	2.0	4.6	—	105	2.2	5.5	5.2	6.6	−0.2	−0.9	+0.3	—
8	68	$\underline{3\vert}$	1.6	3.5	4.1	112	2.8	3.5	—	—	−1.2	0.0	—	—
9	68	$\vert\underline{2}$	2.1	4.0	5.0	112	2.8	2.8	—	—	−0.7	+1.2	—	—
10	55	$\underline{2\vert}$	1.0	2.7	4.0	122	0.8	2.6	2.5	3.9	+0.2	+0.1	+0.1	+0.1
11	34	$\underline{3\vert}$	1.1	3.5	4.5	138	1.6	4.0	3.8	4.7	−0.5	−0.5	+0.2	−0.2
12	37	$\underline{2\vert}$	1.6	2.8	4.0	144	1.6	2.6	3.0	4.6	0.0	+0.2	−0.4	−0.6
13	57	$\underline{3\vert}$	0.7	2.8	4.5	145	1.4	2.5	2.8	4.5	−0.7	+0.3	−0.3	0.0
14	64	$\underline{2\vert}$	1.4	3.0	4.5	162	1.9	3.5	—	—	−0.5	−0.5	—	—
15	50	$\underline{2\vert}$	1.3	3.5	4.5	196	1.0	3.3	3.2	5.3	+0.3	+0.2	+0.1	−0.8

A good example of complete healing following the flap operation is seen in Case 2 (Fig. 4, *A*). The new epithelial attachment is on enamel, slightly coronal to the old cementoenamel junction. Actually, under high-power magnification, a narrow zone (0.1 mm.) of new cementum can be seen extending from the old cementum to cover adjacent enamel (Fig. 4, *B*). The explanation for this must be that the bottom of the epithelial attachment was severed during the flap operation and connective tissue came in contact with the enamel when the flap was sutured back. Before the epithelial attachment reached the old cementum, new cementum was formed and a gingival fiber attachment to the enamel was established. This is of academic interest rather than of practical or clinical interest because of the rarity of occurrence of such an attachment and because of the very short distance involved.

A.



B.

Fig. 4.—Case 2. *Top*, Complete healing twenty-eight days after the flap operation. (Magnification, ×11; reduced 1/6.)

Bottom: High magnification of the cementoenamel junction seen in the upper photomicrogram. New cementum forming and gingival fibers being attached from *A* to *B* on the surface of the enamel. Old cementum slightly overlapping the enamel. (Magnification, ×400; reduced 1/6.)

Another example of good healing is seen in Case 4 (Fig. 5, *A* and *B*). The new epithelial attachment here ends 0.1 mm. apical to the cementoenamel junction. In Case 5 (Fig. 6) there is evidence of slight resorption and repair of the cementum at the bottom of the new epithelial attachment. This is the only instance in which any resorption of the cementum, related to the surgical flaps, could be observed.

Residual foreign bodies of various types were found within the healed site of the previous surgical separation in six of the fifteen cases. It must

A.

B.

Fig. 5.—Case 4. *A*, Complete connective tissue healing almost to the cementoenamel junction. Small piece of supragingival calculus, but very thin epithelial attachment in the cervical region. (See clinical photographs, Fig. 2, *A* and *B*.)

B, High magnification of the cementoenamel junction from *A*. The epithelial attachment extends only 0.1 mm. down on cementum. (Magnification, ×357; reduced ⅙.)

Fig. 6.—Case 5. Slight resorption and beginning repair of the cementum at the bottom of the epithelial attachment. (Magnification, ×459; reduced ⅙.)

be assumed that these foreign bodies had been implanted at the time of the flap operation. The foreign bodies consisted of calculus, tooth fragments, and cotton material (probably from surgical gauze sponges). The foreign bodies were small, and they seemingly had not interfered with the general healing of the flap to the tooth and the alveolar process. Remains of calculus were observed in Cases 7 and 9 and probably in Case 10. These patients had calculus on their teeth at the time of the flap operation, and no attempt was made to remove the calculus prior to surgery. The other patients had very little or no calculus on the teeth that were included in the experiment.

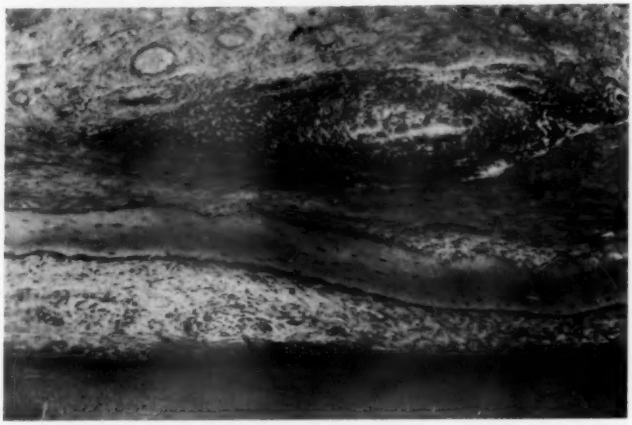


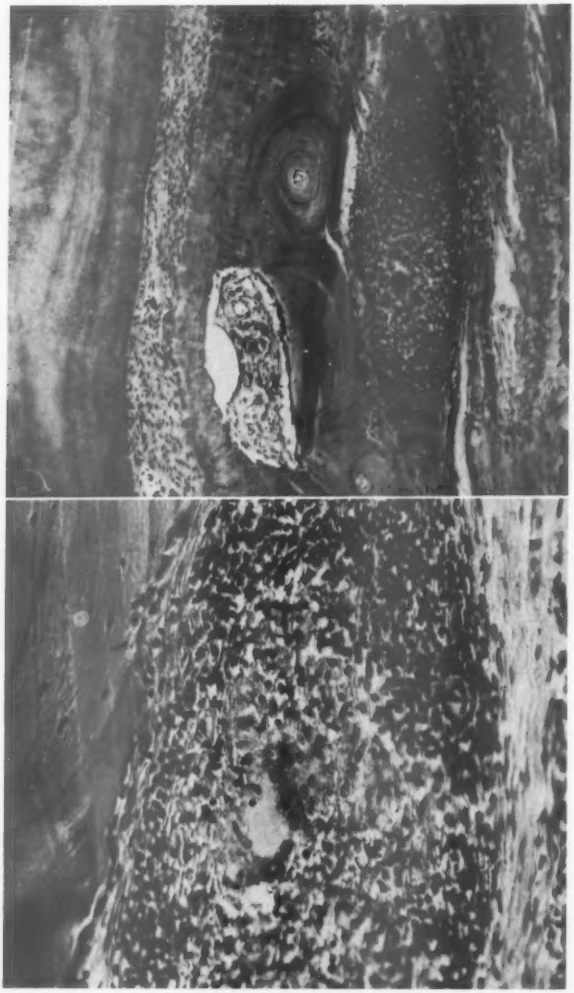
Fig. 7.—Case 9. Focal area of chronic inflammation and foreign body giant-cell reaction on the surface of the periosteum, 7 mm. apically to the free gingival margin. When observed under the microscope, the foreign material strongly resembled calculus. (Magnification, ×256; reduced ⅙.)

The severe chronic inflammation and foreign body reaction to calculus seen in Fig. 7 (from Case 9) was located about 7 mm. apical to the free gingival margin on the surface of the periosteum on the labial aspect of the alveolar process. This specimen was removed 112 days following flap operation, so it apparently takes a relatively long time for the body to eliminate implanted calculus. The foreign material seen in Fig. 8, *A* and *B* (from Case 10) could not be definitely identified, but it probably also represents calculus. The reaction in this 122-day specimen is very similar to that seen in Fig. 7 (Case 9), where the morphology of calculus still can be recognized.

Cotton material embedded in the periosteum is seen in Fig. 9, *A*, and cotton almost surrounded by bone is seen in Fig. 9, *B*. The reaction to the cotton is very mild; only a few foreign body giant cells and a few inflammatory cells are present.

A few tooth fragments were observed in the zone of healing (Fig. 10). These fragments must have originated from adjacent teeth when these teeth were extracted in conjunction with the flap operation. The tooth fragments were surrounded by connective tissue, and they elicited a very mild reaction as previously reported by Ramfjord[10] and Schaffer.[11]

A.

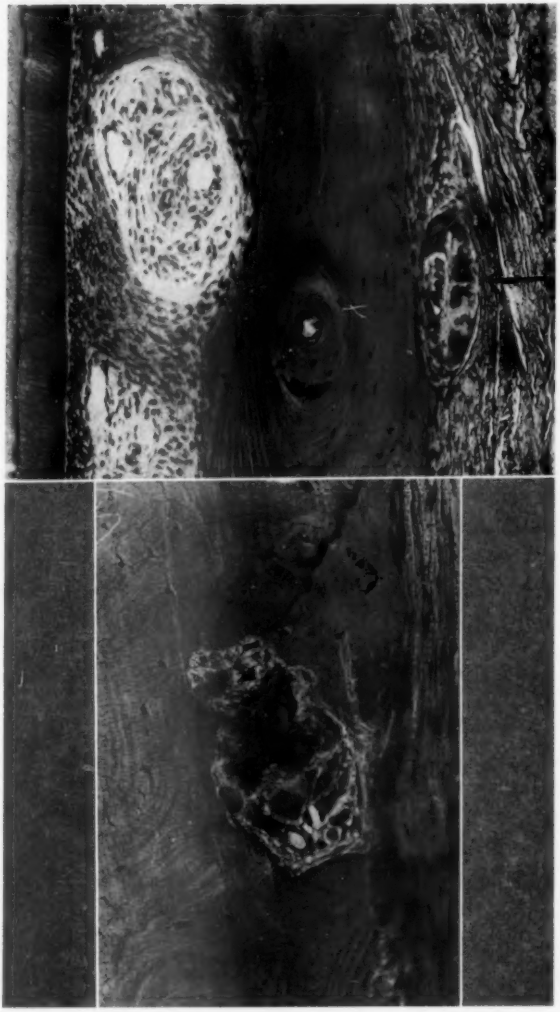


B.

Fig. 8.—Case 10. *A*, Focal area of chronic inflammation and foreign body reaction in the periosteum 3 mm. apical to the alveolar crest. (Magnification, ×140; reduced ⅙.)

B, High magnification of the central zone of the inflammatory focus shown in *A*. Note foreign body giant cells. When the microscope was focused up and down, the foreign bodies appeared as fragments of calculus. (Magnification, ×600; reduced ⅙.)

A.



B.

Fig. 9.—*A*, Case 4. Foreign body giant-cell reaction to cotton within the periosteum on the labial surface of the alveolar process. (Magnification, ×257; reduced ⅙.)

B, Case 13. Foreign body giant-cell reaction to cotton in bony notch on the labial surface of the alveolar process. (Magnification, ×600; reduced ⅙.)

No attempt was made to determine whether or not bacteria were present in the areas of foreign body reaction. At least it is very likely that the residual fragments of embedded calculus harbored microorganisms and that they might act as foci of infection.

The presence of foreign material within the zone of the previous surgery in six out of fifteen gingival mucoperiosteal flaps should emphasize the need for careful surgical procedures. Calculus should be completely removed routinely prior to the flap procedure. Suction should be used in preference to surgical gauze sponges. Irrigation and adequate aspiration beneath the flap are important whenever bone or tooth fragments have been loosened while the flap was deflected.

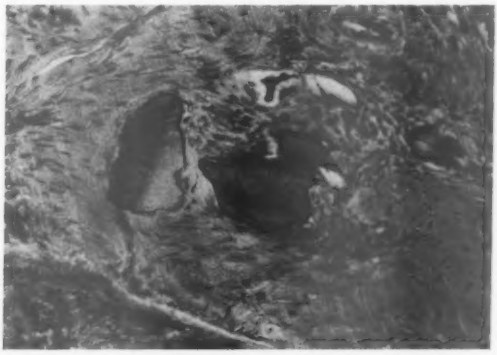


Fig. 10.—Case 15. Fragments of cementum and dentine within the site of the previous flap operation. Slight osteoclastic activity and mild chronic inflammation is some areas. Around one fragment there is no reaction. (Magnification, ×280; reduced ⅙.)

SUMMARY

1. Surgical mucoperiosteal flaps separating the gingiva from the teeth healed without any significant loss of periodontal attachment in all of the fifteen cases examined.

2. Presence or absence of presurgical inflammation or variation in occlusal forces did not seem to influence the healing.

3. By means of serial sectioning, it was possible to observe foreign bodies in the zones of previous surgery in six of the fifteen cases. Focal chronic inflammation was rather severe in three of these six cases three to four months after the flap procedure.

4. Calculus should be removed routinely prior to flap operations, and suction should be used rather than sponges during the operation in order to avoid implantation of foreign bodies.

Acknowledgment is made to Drs. G. Kiester and E. R. Costich for assistance with the surgical removal of several of the specimens.

REFERENCES

1. Borden, S. M.: Histological Study of Healing Following Detachment of Tissues as is Commonly Carried Out in the Vertical Incision for the Surgical Removal of Teeth, J. Canad. Dent. A. **14**: 510, 1948.
2. Dingman, R. O., and Hayward, J. R.: Oral Surgery in General Practice, J. Am. Dent. A. **35**: 610, 1947.
3. Fish, E. W.: Parodontal Disease; a Manual of Treatment and Atlas of Pathology, London, 1946, Eyre & Spottiswoode, Ltd.
4. Glickman, I., and Lazansky, J. P.: Reattachment of the Marginal Gingiva and Periodontal Membrane in Experimental Animals, J. D. Res. **29**: 659, 1950.
5. Irwin, D. H.: A Histological Study of the Area of Attachment of Gingival Tissue Following Its Surgical Detachment, Thesis, Northwestern University, 1953.
6. Linghorne, W. J., and O'Connell, D. C.: Studies in the Regeneration and Reattachment of Supporting Structures of the Teeth, J. D. Res. **30**: 604, 1951.
7. Linghorne, W. J., and O'Connell, D. C.: Studies in the Regeneration and Reattachment of Supporting Structures of the Teeth, J. D. Res. **34**: 164, 1955.
8. Morris, M. L.: The Reattachment of Human Periodontal Tissues Following Surgical Detachment, J. Periodont. **24**: 220, 1953.
9. Morris, M. L.: Healing of Human Periodontal Tissues Following Surgical Detachment From Non-Vital Teeth, J. Periodont. **28**: 222, 1957.
10. Ramfjord, S.: Experimental Periodontal Reattachment in Rhesus Monkeys, J. Periodont. **22**: 67, 1951.
11. Schaffer, E. M.: Cementum and Dentine Implants in a Dog and a Rhesus Monkey, J. Periodont. **28**: 125, 1957.
12. Skillen, W. G., and Lundquist, G. R.: An Experimental Study of Periodontal Membrane Reattachment in Healthy and Pathologic Tissue, Dental Cosmos **24**: 175, 1937.
13. Stones, H. H.: The Reaction and Regeneration of Cementum in Various Pathological Conditions, Proc. Roy. Soc. Med. **27**: 728, 1934.
14. Waerhaug, J.: The Gingival Pocket, Odontol. Tidskr. **60**: Supp. 1, 1952.
15. Workman, G. N.: Studies in Reattachment of the Gingiva, Thesis, Northwestern University, 1947.

Section by the

ARMED FORCES AND PUBLIC HEALTH SERVICE

ORAL MANIFESTATIONS OF MACROGLOBULINEMIA OF WALDENSTRÖM

Report of a Case

Jack W. Gamble, D.D.S. and Edward J. Driscoll, D.D.S.,** Bethesda, Md.*

INTRODUCTION

MACROGLOBULINEMIA was first described as a clinical entity by Waldenström in 1944.[1] He reported three cases in which the presence of an increased amount of serum macroglobulin was demonstrated by ultracentrifugal analysis. Clinical findings were fatigue, epistaxis, gingival hemorrhage, and a moderate lymphadenopathy. There were an anemia, an elevated sedimentation rate, a bone marrow lymphocytosis, and a hyperglobulinemia.

Voigt and Frick[2] reported a case in which the chief complaints were epistaxis, weakness, weight loss, and persistent bleeding from the site of a recently extracted tooth. Hanlon, Bayrd, and Kearns[3] reported four cases. In their second case the chief complaint was profuse bleeding following the extraction of a tooth.

In his classical review of the literature in 1958,[4] Waldenström noted approximately 100 cases of macroglobulinemia in the medical literature. The disease was rare in persons under 40 years of age, and the incidence was 2.2 times greater in males than in females. The disease was most prevalent in the 50- to 70-year age group. In a high percentage of his cases, epistaxis and gingival hemorrhages as well as disturbances of vision were early signs. Some of the cases had salivary gland involvement with an accompanying xerostomia.

Recently a detailed histopathologic study of three cases was reported by Dutcher and Fahey.[5] They stated that the disease is probably a neoplastic

---

This case is part of a study conducted by Dr. John L. Fahey of the National Cancer Institute, National Institutes of Health, United States Public Health Service, Department of Health, Education, and Welfare.

*Senior Assistant Dental Surgeon, United States Public Health Service, Clinical Center, National Institutes of Health, Department of Health, Education, and Welfare.

** Chief of Oral Surgery Section, National Institute of Dental Research, National Institutes of Health, United States Public Health Service, Department of Health, Education, and Welfare.

proliferation of reticuloendothelial origin and that the only certain method of establishing the diagnosis is the demonstration of increased serum macroglobulins by ultracentrifugal analysis. The oral manifestations of Dutcher and Fahey's second case are presented here to familiarize the dental profession with this disease.

The oral condition was particularly interesting in this case because of the nature of the ulcerations. Even though ulcerations have been previously reported in this disease, none were of the size, depth, and character as those in this patient. It seems probable that the course and the severity of the lesions in this case were influenced by the injudicious use of Aspergum.

CASE REPORT

History of Present Illness.—Patient J. W., a 43-year-old white man, was in good health until September, 1956, at which time he noticed the passage of several tarry stools within a period of twenty-four hours.

In November, 1956, following a semiannual dental examination and prophylaxis, the patient hemorrhaged severely from the gingivae for three days. In March, 1957, the patient returned to his dentist, and at this time another prophylaxis was performed. The dentist was unable to control the profuse gingival bleeding, and the patient was referred to an oral surgeon who used electrocautery without success. The patient was then hospitalized by his physician, and in the following days he was given 4 units of fibrinogen (2.7 gram per unit) and four transfusions (500 ml. each) of whole blood. Local oral procedures, such as the use of topical thrombin and pressure, were employed. The hemorrhage was arrested in approximately seventeen days. It was during this period of hospitalization and follow-up that the diagnosis of macroglobulinemia was made after a bone marrow study revealed abnormal plasma cells and serum ultracentrifugation disclosed abnormal macroglobulins.*

The laboratory findings at this time were as follows: red blood cells, 3,100,000; hemogloblin, 10.4 Gm. per cent; HCT, 30 per cent; white blood cells, 5,250 with a normal differential; prothrombin time, 51 per cent of normal; and total serum protein, 10.3 Gm. per cent of which 5.6 Gm. per cent was globulin. (Normal serum protein = 6.0 to 7.5 Gm. per cent; globulin, 2.5 to 3.0 Gm. per cent.)

In April, 1957, the gingival, buccal, and pharyngeal mucosae were growing progressively tender. Toward the latter part of July, 1957, small punched-out ulcerations appeared on the tongue, buccal, and soft palatal mucosae. These areas, along with the gingival tissues, hemorrhaged frequently and were painful.

In the early part of September, 1957, after having tried anesthetic lozenges for pain associated with the lesions with no beneficial results, the patient began to chew ten to twenty tablets of Aspergum daily. Each of these tablets contained 0.2 Gm. of aspirin. The patient placed the gum on his teeth to protect the tongue ulcerations, and into the other ulcerations to stop the hemorrhage.

The patient was admitted to the Clinical Center, National Institutes of Health, on Oct. 2, 1957. At this time a review of past and family history, other than that already presented, was noncontributory.

Physical Examination.—The pertinent findings were generalized lymphadenopathy, hepatosplenomegaly, bleeding oral ulcers, a hyperemic bowel which oozed blood on slight pressure, and retinal hemorrhages.

The first laboratory reports at the Clinical Center were as follows: red blood cells, 2,273,000; hemoglobin, 8.2 Gm. per cent; HCT, 24 per cent; reticulocytes, 0.5 per cent;

*The diagnosis was made by Drs. Edward Adelson, Washington, D. C., and Norman Shoemaker, Silver Spring, Maryland.

white blood cells, 6,800 with a normal differential; platelets, 200,000; erythrocyte sedimentation rate, 2 mm. per hour. Serology and liver function tests were normal except for a serum protein of 12.9 Gm. per cent, of which 2.2 Gm. per cent was albumin and 10.7 Gm. per cent was globulin. Most of the increase in serum protein was due to macroglobulins ($S_{20w}^{o} = 18S$) which migrated electrophoretically as gamma globulin. The sternal marrow was normal except for a number of lymphoid plasma cells. Urinalysis was normal.

Dental Examination.—The patient was referred to the dental clinic, Clinical Center, National Institutes of Health, with the chief complaint of extreme tenderness and pain in the oral cavity.

Punched-out, discrete ulcerations (Fig. 1) were noted bilaterally on the tongue; each measured approximately 1 cm. Bilaterally, on the buccal mucosa opposite the molars and to the left of the midline in the soft palate, slightly smaller ulcerations were seen. The buccal and pharyngeal mucosae, gingivae, and uvula were edematous, red, and painful. The ulcerations were free of any exudate. The buccal and gingival mucosae oozed blood freely, and the teeth were very mobile.

Fig. 1.—Tongue lesion when patient was first seen at the Clinical Center.

Treatment.—There was some concern as to the possibility of an associated oral fungal infection (monilia), and for this reason Mycostatin therapy was instituted on Oct. 7, 1957. One million units were given orally every six hours, and an oral suspension was advised as a mouthwash. Meticorten, 25 mg. orally every six hours, was started on October 12 as treatment for the macroglobulinemia. The patient was advised to avoid using Aspergum, which he chewed and packed into the ulcerations. Although it was producing analgesia, there was a strong possibility that the salicylic acid was aggravating the already damaged tissues.

Multiple lymph nodes were biopsied on October 10 and 11, and the patient had some difficulty with postoperative hemorrhage so he remained in bed. He was seen on the ward by the dental officer until October 16, at which time there was less pain and tenderness in the mouth and no hemorrhage was noted.

On October 21 there was general improvement of symptoms and the Meticorten was reduced to 40 mg. per day. The patient was still using Aspergum, against advice, but in reduced amounts (five to seven tablets daily). Upon reduction of the Meticorten, the lesions again became more painful and the patient increased the use of Aspergum until November 5, when he was chewing an average of ten tablets daily. Despite the increased use of the gum, the lesions were causing considerable pain and distress. Dyclonine 0.5

per cent, 4 ml. as needed, was prescribed as a mouth rinse. This antihistamine had been used beneficially at the Clinical Center to relieve pain associated with aphthous ulcers. The patient became quite upset on the ward and stated that he was experiencing no relief of pain. Beginning November 6, he was given Aspergum at his bedside by the physician and was told to use it sparingly.

An impression was made of the mandibular teeth, and a clear acrylic splint was constructed and delivered to the patient on November 7 (Fig. 2). The patient stated that the tongue lesions were much more comfortable with the splint in place.*

On November 8 the Meticorten was reduced to 20 mg. daily. The lesions again became painful, and the patient began using twelve to sixteen tablets of Aspergum daily. On November 14 the ulcerations were still very painful, and they were increasing in size.

Fig. 2.—Clear acrylic splint used to cover lower teeth and prevent irritation to the lingual ulcerations. When photographed, the splint appears metallic.

At this time additional bacteriologic and cytologic smears were taken, but these were not revealing. The dyclonine was discontinued, and Xylocaine viscous, 5 ml. as needed, was started as a mouth rinse. Procaine penicillin, 600,000 units intramuscularly every twelve hours, was started. This was changed for convenience in nine days to benzanthine penicillin G, 1,200,000 units intramuscularly. The lesions on the soft palate and on the right and left sides of the tongue were increasing in size, and on November 23 they were large and deep but unusually clean (Figs. 3 and 4).

There had been some improvement in the patient's systemic symptomatology, and he was allowed to go on a two-day pass. The patient returned to the hospital on November 25, at which time the oral lesions were continuing to degenerate. It was learned that the patient was still using Aspergum, placing the gum in the ulcerations before he retired at night. There was concern about the size and depth of the lesions and fear that an arterial hemorrhage might occur, particularly in the lingual areas. A vigorous regimen was instituted to force the patient to discontinue the use of Aspergum. Meprobamate, 2 Gm. daily in divided doses, and codeine sulfate were prescribed for pain, and gentian violet 1 per cent was applied locally.

Two days later at 2 A.M., the patient awoke with his mouth filled with blood. Oozing continued from the lesion on the left side of the tongue, despite pressure applications, until 7:30 A.M. when it stopped spontaneously.

*Splint constructed by and on the advice of Dr. Ralph S. Lloyd, Chief of Dental Department, Clinical Center.

The patient's general condition began to improve on Meticorten and penicillin therapy. The liver and spleen were no longer palpable, the anemia was greatly improved, and the retinal hemorrhages had resolved. The patient at this time had finally yielded to the opinion that the Aspergum was influencing his oral condition adversely. Several days after he discontinued the gum, the lesions began to respond favorably.

Fig. 3.—Lingual ulceration on Nov. 23, 1958, when it was most severe.

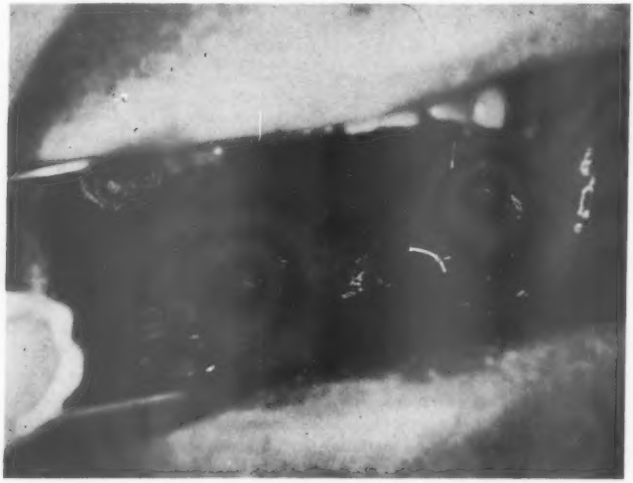


Fig. 4.—Soft palatal ulceration on Nov. 23, 1958.

On December 2 his systemic and oral condition was sufficiently improved for his discharge to outpatient status. He was seen weekly, and on each visit a gentle maintenance prophylaxis was performed.

The penicillin was discontinued on Jan. 20, 1958, and the patient was maintained on 20 mg. of Meticorten daily.

The patient was readmitted to the hospital on Feb. 8, 1958, with deterioration of his general condition. The liver and spleen were again palpable, and new retinal hemorrhages were noted. The hemoglobin had fallen to 8.8 Gm. per cent with an HCT of 28 per cent and a normal white and differential count.

Oral examination revealed continued healing of the ulcerations, but the gingivae palatal to the maxillary anterior teeth were red and edematous and oozed small amounts of blood. There was cervical lymphadenopathy.

Urethane, 0.02 Gm. per kilogram of body weight per day, was started on Feb. 13, 1958, for treatment of the macroglobulinemia. The patient improved symptomatically until February 20, at which time he was again discharged to the outpatient department. The Meticorten was reduced gradually and was discontinued on February 23.

The patient was seen bi-weekly in the dental clinic, at which times the mouth was treated by irrigation and light supra- and subgingival scaling.

Fig. 5.—Lingual lesion completely healed as shown in April, 1959.

On April 21 a gingival biopsy was done between the upper left cuspid and first premolar to see if lymphoid plasma cells could be found in the gingivae. Special stains were employed, but no significant histopathologic findings were noted with the exception of chronic inflammation.

The urethane was discontinued on June 5, after having been gradually increased to 0.1 Gm. per kilogram of body weight per day. The patient again was symptomatically improved, although there had been no change in the serum proteins.

He was seen at weekly intervals following the cessation of urethane therapy until July, 1958. He is now being seen at monthly intervals, at which times a careful prophylaxis is done. The teeth have become less mobile, and the patient has continued to be free of oral symptoms with the exception of slight bleeding when he brushes his teeth. The ulcerations have completely healed and in April, 1959, there was only a faint indication of their previous location (Fig. 5).

DISCUSSION

A case of macroglobulinemia of Waldenström is presented. This patient had peculiar deep and painful ulcerations of the oral mucosa in addition to gingival hemorrhages. There have been no such cases reported in the dental literature, so far as could be determined.

The oral ulceration preceded the use of the Aspergum by almost two months, so it is probable that these lesions were originally oral manifestations of this disease. The effect of the Aspergum on their course and severity must be considered.

The etiology of the oral ulcerations and hemorrhage is unknown. As for the latter, the bleeding time, coagulation time, prothrombin time, prothrombin activity, and serum fibrinogen fluctuate greatly in these patients.[4] The reader is referred to the medical literature[2, 6-10] regarding the hemorrhage associated with this condition.

Any oral surgical procedure and any deep scaling in a patient with macroglobulinemia is to be approached with extreme caution. We are of the opinion that, dentally, these patients should be treated like patients with leukemia. For a regimen of this type of care, the reader is referred to a recent report in ORAL SURGERY, ORAL MEDICINE AND ORAL PATHOLOGY.[11]

REFERENCES

1. Waldenström, J.: Incipient Myelomatosis or Essential Hyperglobulinemia With Fibrinogenopenia: A New Syndrome? Acta med. scandinav. **117:** 216-242, 1944.
2. Voigt, A. E., and Frick, P. G.: Macroglobulinemia of Waldenström: A Review of the Literature and Presentation of a Case, Ann. Int. Med. **44:** 419-425, 1956.
3. Hanlon, D. G., Bayrd, E. P., and Kearns, T. P.: Macroglobulinemia; Report of Four Cases, J. A. M. A. **167:** 1817-1821, 1958.
4. Waldenström, J.: Die Makroglobulinaemie; Ergebnisse der inneren Medizin und Kinderheilkunde—Neue Folge 9. Band. 586-621, 1958 (Translation).
5. Dutcher, T. F., and Fahey, J. L.: The Histopathology of the Macroglobulinemia of Waldenström, J. Nat. Cancer Inst. **22:** 887-917, 1959.
6. Laurin, J. G., Beauregard, J. M., and Provost, G.: Waldenström's Purpura Hyperglobulinemia Associated With Macroglobulinemia, Canad. M. A. J. **76:** 1957.
7. Long, L. A., Riopelle, J. L., Francoeur, M., Pare, A., Poirier, P., Georgeso, M., and Colpron, G.: Macroglobulinemia: Effect of Macroglobulinemia on Prothrombin Conversion Accelerators, Canad. M. A. J. **73:** 726-733, 1955.
8. Jim, R. T., and Steinkamp, R. C.: Macroglobulinemia and Its Relationship to Other Paraproteins; With a Case Report, J. Lab. & Clin. Med. **47:** 540-561, 1956.
9. Strfanini, M.: Bull. New York Acad. Med. **30:** 239, 1954.
10. Tischenforf, W., and Hartman, F.: Makroglobulinaemie (Waldenström) mit gleichzeitiger Hyperplasia, Acta. haematol. **4:** 374, 1950.
11. Duffy, J. H., and Driscoll, E. J.: Oral Manifestations of Leukemia, ORAL SURG., ORAL MED. & ORAL PATH. **11:** 5, 1958.

MULTIPLE HEREDITARY FAMILIAL EPITHELIAL CYSTS OF THE JAWS WITH THE ASSOCIATED ANOMALY OF TRICHOEPITHELIOMA

Report of a Case

Lowell E. McKelvey, B.A., D.D.S., M.S., Fort Sam Houston, Texas,*
*Cecil R. Albright, B.S., D.D.S., M.S.,** Fort Bragg, N. C., and*
*George Prazak, M.D.,*** Fort Sam Houston, Texas*

THE case reported here is considered a rare and interesting one, particularly since the occurrence of multiple epithelial cysts of the jaws was linked with the concurrent anomaly of trichoepithelioma (referred to in earlier literature as epithelioma adenoides cysticum). Only brief mention has been made of the associated congenital defects, especially those of a hereditary or familial nature, that sometimes accompany trichoepithelioma.

According to a summary of the literature made by Summerill and Hutton[9] in 1932, the earliest recorded case of multiple benign cystic epithelioma was that reported by Ancell in 1841. In articles by White,[11] Brooke,[3] Fordyce,[4] and Savatard,[8] no references were made to a concurrent finding of cysts of the jaws. Binkley and Johnson[2] were the first to report a case of multiple dental cysts in the jaws of a patient with epithelioma adenoides cysticum. At the time of their writing in 1951, they had not found any similar case mentioned in the literature.

Gross,[5] in 1953, reported the case of a 9-year-old white boy with lesions of trichoepithelioma plus odontogenic cysts of the mandible and maxilla. This patient's father, mother, and one 6-year-old brother were living and well. There was no evidence of skin lesions in the father, mother, or brother. No mention was made of the presence or absence of cystic lesions of the jaws in other members of the family.

As suggested by Summerill and Hutton[9] and supported by others, trichoepithelioma is often hereditary; it is a simple dominant characteristic that affects two generations in succession but rarely passes to the third. It is a comparatively rare disease of the skin, which begins in youth or infancy and most

*Chief, Oral Surgery, Brooke Army Hospital.
**Chief, Oral Surgery, Womack Army Hospital.
***Chief, Dermatology, Brooke Army Hospital.

often affects the female. Clinically, it is characterized by multiple small, papular, firm, benign lesions originating from the basal cells of the squamous epithelium or hair follicles.

The literature contains numerous references to multiple cysts of the jaws in one person. However, reports of evidence of hereditary or familial background in the occurrence of cysts of the jaws are rather sparse. As mentioned by Thoma and Blumenthal,[10] undoubtedly more cases of these conditions have been observed than have been recognized and recorded. Perhaps a more thorough inquiry into the family history of patients with epithelial cysts of the jaws would bring to light increased numbers of such occurrences. Thoma and Blumenthal's genealogic chart of a family with cysts supports the comments of others that in some cases the occurrence of cysts in the jaws may have a familial or hereditary background. In this family history, the father had one cyst and the mother had none. This family was followed through the three succeeding generations. There were thirty-two progeny, of whom fourteen had one to six cysts in the jaws, four were not available for examination, and the rest were without cystic lesions.

Beyrent[1] reported finding multiple cysts of the jaws in four siblings of one family. The other siblings and the parents exhibited no similar lesions. Few other such references have been found.

The character, distribution, and specific features of the lesions in the case to be presented here are not to be confused with the condition reported by Jones[6] as familial multilocular cystic disease of the jaws or "cherubism." Our case does parallel the familial or hereditary tendency manifest in the case reported by Thoma and Blumenthal[10] with the additional findings of the lesions of trichoepithelioma as reported by Binkley and Johnson[2] and Gross.[5]

CASE REPORT

On Sept. 14, 1955, a white male soldier, 21 years of age, was admitted to Brooke Army Hospital for evaluation and treatment of multiple cysts of the maxillae and mandible.

Examination.—The patient, a tall slender, asthenic person, was alert, cooperative, and in no [illegible] distress. His temperature was 98.6° F. His blood pressure was 130/72 mm. Hg, and he had a pulse rate of 86. There were no unusual findings except for the lesions described below. Several small superficial sebaceous-like cysts were present on the upper eyelids. Numerous translucent, shiny, rounded, papular lesions, many of which were pigmented, were noted on the eyelids, nose, nasolabial folds, and forehead, behind the ears, and on the chest (Fig. 1). Examination of the mouth revealed edentulous, somewhat atropic alveolar ridges with a normal-appearing mucosal cover. Roentgenographic examination disclosed unilocular areas in all four quadrants of the maxillae and mandible, with no evidence of retained roots or impacted teeth. The routine chest roentgenogram was negative.

History of Present Illness.—This history of cystic lesions of the jaws dated back approximately seven years to the finding and removal of a cyst in the right maxilla by the patient's dentist. Three years later "five or six" cysts were removed from the left maxilla and the mandible, bilaterally. Since that time the patient had no signs or symptoms of lesions of the jaw until he was informed of the x-ray findings after entering military service (Fig. 2). Two years earlier he had been treated by an ophthalmologist for a cystic lesion of the right upper eyelid. Cystic lesions of the left upper lid were removed on three occasions. About eight months before he was admitted to Brooke Army Hospital he noted the development of a cystic lesion of the left upper eyelid, but he did not seek treatment.

Fig. 1.—Skin lesions of the face characteristic of trichoepithelioma.

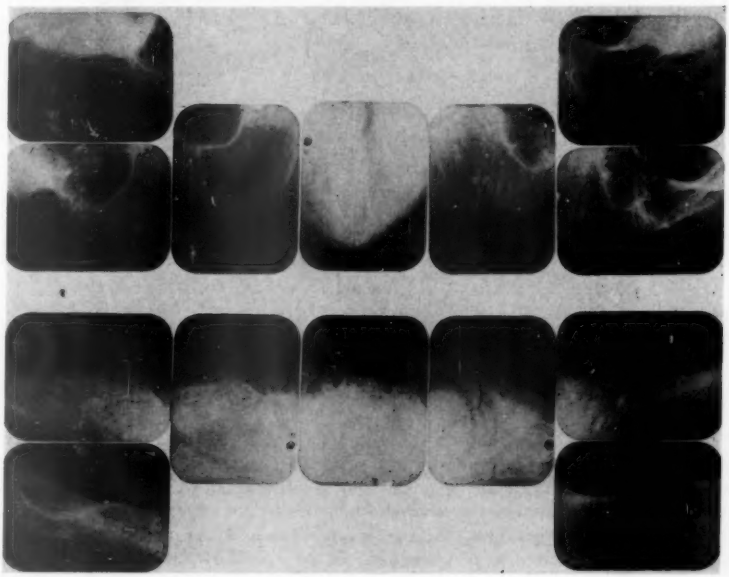


Fig. 2.—Multiple unilocular cystic areas in the bony arches of the maxillae and mandible.

For about the last ten years the patient had noted numerous small nodular lesions on the skin of the ears, nose, and anterior chest. A lesion removed from behind the right ear several years ago was reported as "basal-cell carcinoma."

Past History.—Other than the information contained in the history of the present illness, there apparently had been no contributory past history.

Family History.—The patient's mother and father were living and were apparently in good health, aside from the fact that the father had similar cystic lesions in the jaws which were removed in 1952 by a civilian oral surgeon. The patient was the second of three male siblings, all of whom had had cystic lesions in the jaws. A 27-year-old brother had had multiple cysts in all quadrants of his jaws, the first when he was 17 years of age. He, too, was treated by a civilian oral surgeon, who surgically removed all the cysts and deemed it advisable to sacrifice all the patient's teeth. Even after these extractions were accomplished, some of the cystic lesions were said to have recurred. A 17-year-old brother had had multiple cystic lesions in all quadrants of his jaws, the first when he was 15 years old. His cysts were removed in a local civilian hospital, and numerous teeth were extracted at the time of surgery. To date there had been no evidence of any recurrence in his case. The patient's three sisters, aged 9, 18, and 23 years, were apparently in good health and had no history of any cystic lesions in the maxillae or mandible. The remainder of the family history was noncontributory.

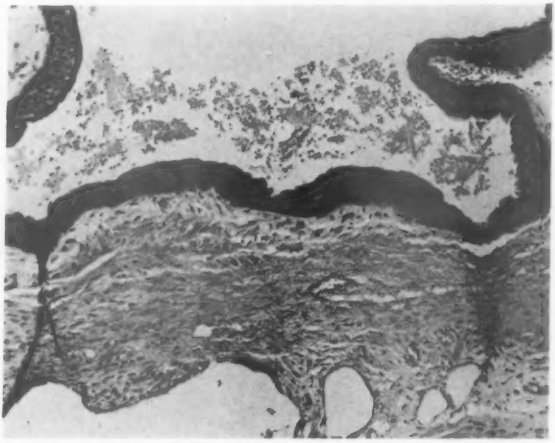


Fig. 3.—Photomicrograph of a segment of one of the representative bone cysts. Note the heavy fibrous wall and well-developed epithelial lining of the lesion. (Magnification, ×105; reduced ¼.)

Laboratory Findings.—The routine hematology, urinalysis, and microflocculation tests were within normal limits.

Treatment and Course.—On Sept. 15, 1955, the cysts in the right maxilla were surgically removed under local anesthesia, and the specimen was sent to the pathology laboratory for microscopic examination. The resulting wound was saucerized and packed open with a Peruvian balsam drain, which was changed at frequent intervals. The cystic areas in the other three quadrants of the mouth were operated upon and treated in a like manner. The cyst in the left upper quadrant was fixed to the membrane of the maxillary antrum. This small attachment of the antral membrane was excised to eliminate a possible nidus for recurrence of the cyst. All the laboratory reports on the cystic lesions noted them to be benign, epithelial cysts with some fibrotic changes (Fig. 3). Each of the resultant wounds healed normally without complication.

On Sept. 15, 1955, the patient was referred to the eye clinic for evaluation and treatment of the cystic lesions of the eyelids. These were diagnosed as an infected chalazion of the left upper eyelid and minute noninfected chalazions of the right upper eyelid. Several were removed on Oct. 17, 1955. The pigmented lesion in the right retroauricular area was surgically excised by the plastic surgery service and sent to the pathology laboratory for microscopic examination. On Oct. 24, 1955, this specimen was reported as a pigmented, basal-cell epithelioma which appeared to be adequately excised (Fig. 4). On Nov. 3, 1955, the remaining chalazions were removed.

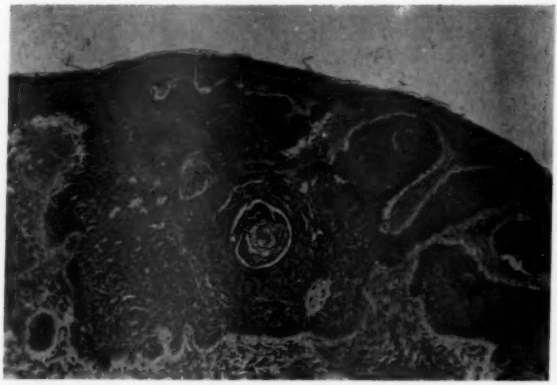


Fig. 4.—Photomicrograph of one of the lesions diagnosed as pigmented basal-cell nevus; typical basal-cell proliferation with well-circumscribed palisaded perimeter. (Magnification, ×105; reduced ¼.)

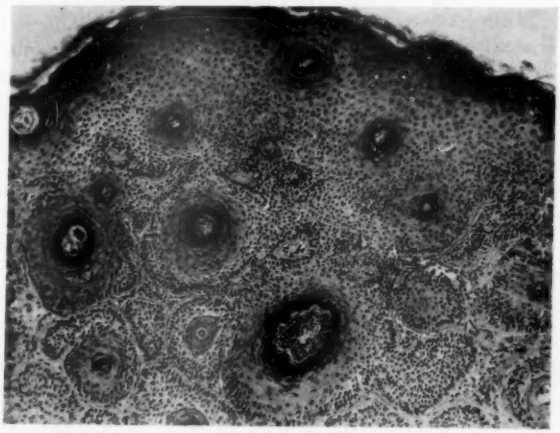


Fig. 5.—Photomicrograph of one of the lesions diagnosed as trichoepithelioma. The prominent features of this lesion are the multiple islands of basal cells, usually with a progression from the orderly peripheral cell layers to a keratin or rudimentary hair-shaft center. (Magnification, ×105; reduced ¼.)

The patient was referred to the dermatology clinic for evaluation and treatment of the multiple lesions of the face and chest. These lesions were diagnosed as trichoepithelioma, proved by biopsy (Fig. 5). The dermatology clinic chose to treat for these lesions on an

outpatient basis following the patient's discharge from the hospital. However, elevated, pigmented nodules were noted on the right pectoral region, and a punch biopsy of one of them was performed on Nov. 4, 1955. This nodule was subsequently reported by the pathology laboratory as a basal-cell epithelioma, adequately excised.

All the biopsied sites healed normally, and the patient was discharged from Brooke Army Hospital to duty on Nov. 16, 1955.

DISCUSSION

The patient has been advised to have follow-up examinations of the jaws and skin lesions at yearly intervals, or more often if conditions so indicate. At the first twelve-month check-up there was no evidence of recurrence of the cystic lesions of the jaw or of the chalazion sites; nor were there any changes at the sites of removal of the skin lesions on the neck, face, and chest. Some question arises as to whether in the past there had been recurrence of these bone cysts and skin lesions following the prior surgery or whether these so-called new lesions represented multicentric points of new cyst formation. The possibility is that both conditions may have been present in this case. One wonders whether the conditions of trichoepithelioma and multiple cysts of the bony jaws are factors of the same etiological process, common genesis from primary germ cells,[7] or whether they were only coincidental in this case. Since the literature is so scanty concerning these occurrences, there can be little more than conjecture in answer to the questions posed. Certainly, both conditions are characterized by epithelial changes. Perhaps a diagnosis of trichoepithelioma should warrant an x-ray survey of the patient's jaws for possible bone cyst formation. The skin lesions in trichoepithelioma are usually of cosmetic import only, but transition into basal-cell epithelioma occurs occasionally.[7] Although these skin lesions are subject to recurrence in some instances, their adequate removal will usually result in a favorable prognosis. Where multiple bone cysts are found, other members of the patient's family, especially the progeny, should be checked carefully to rule out similar lesions. In the presence of multiple bone cysts, one must always rule out the possibilities of such disease entities as hyperparathyroidism, Hand-Schüller-Christian disease, multiple myeloma, eosinophilic granuloma, and osteomalacia.

REFERENCES

1. Beyrent, J. R.: Multiple Dentigerous Cysts in Four Members of One Family, J. Am. Dent. A. **25**: 623, 1938.
2. Binkley, G. W., and Johnson, H. H., Jr.: Epithelioma Adenoides Cysticum; Basal Cell Nevi, Agenesis of the Corpus Callosum and Dental Cysts, A. M. A. Arch. Dermat. & Syph. **63**: 73, 1951.
3. Brooke, H. G.: Epithelioma Adenoides Cysticum, Brit. J. Dermat. **4**: 268, 1892.
4. Fordyce, J. A.: Multiple Benign Cystic Epitheliomas of the Skin, J. Cutan. & Genito-Urin. Dis. **10**: 459, 1892.
5. Gross, P. P.: Epithelioma Adenoides Cysticum With Follicular Cysts of Maxillae and Mandible: Report of Case, J. Oral Surg. **11**: 160, 1953.
6. Jones, W. A.: Familial Multilocular Cystic Diseases of the Jaws, Am. J. Cancer **17**: 946, 1933.
7. Lever, W. F.: Histopathology of the Skin, ed. 2, Philadelphia, 1954, J. B. Lippincott Company, p. 363.
8. Savatard, L.: Epithelioma Adenoides Cysticum, Brit. J. Dermat. **34**: 381, 1922.
9. Summerill, Frederick, and Hutton, J. G.: Multiple Benign Cystic Epithelioma (Epithelioma Adenoides Cysticum) Arch. Dermat. & Syph. **26**: 854, 1932.
10. Thoma, K. H., and Blumenthal, F. R.: Heredity and Cyst Formation, Am. J. Orthodontics & Oral Surg. **32**: 273, 1946.
11. White, J. C.: Multiple Benign Cystic Epitheliomas of the Skin, J. Cutan. & Genito-Urin. Dis. **12**: 477, 1884.

EFFECTS OF PROLONGED ANTARCTIC ISOLATION ON ORAL AND INTESTINAL BACTERIA

Robert J. Adams, B.S., D.D.S., and William R. Stanmeyer, B.S., D.D.S., New London, Conn.*

INTRODUCTION

CAN bacteria survive the rigors of the Antarctic? The effects of these continuously low subzero temperatures upon bacteria within the human host have long been of interest to the dentist, physician, and bacteriologist. The possible formation by cold weather isolation of an apparently bacteria-free environment, such as that encountered during recent Antarctic expeditions, has been the subject of much discussion, controversy, and speculation. Previous reports[1, 2] indicated that respiratory infections were practically unheard of following a prolonged period of isolation and that reinfection occurred only after the introduction of new bacteria. On this basis, it seemed impossible to explain how a man could contract Vincent's infection or have the process of dental caries continue after a year's isolation and before the introduction of new bacteria to the resident group. What is the answer to this puzzling situation? Medical science has listened to all fantastic rhymes and reasons which returning Antarctic explorers gave during "fireside conversation" about the strange land where everything happens differently.

To determine the effect of prolonged cold on bacteria within the human host, a program was outlined in which changes in quantities of *Lactobacillus acidophilus,* both oral and intestinal strains, were to be studied on a monthly basis throughout the Antarctic year.

METHODS

Subjects for this investigation were thirty-seven men with a mean age of 27.4 years, who, at the beginning of this study, were stationed at Davisville, Rhode Island, awaiting departure for the Antarctic. Base line control studies were carefully conducted by collecting saliva in individual sterile beakers after stimulation by chewing a piece of sterile, bland, weighed paraffin for ten minutes. A dilution of 1:100 was immediately made, the sample was shaken adequately, and 1 c.c. of this solution was pipetted into a sterile

From the U.S. Naval Medical Research Laboratory, U.S. Naval Submarine Base, New London, Connecticut.

*Assistant Professor of Oral Medicine and Surgery, University of Tennessee College of Dentistry.

Petri dish. Then 30 c.c. of tempered Rogosa's[3] lactobacillus selective medium was added to the sample. The pour plates were incubated at 37° C. for seventy-two hours. The procedure was repeated on three separate days to give a base line control of three separate counts. These thirty-seven men were subsequently transferred to Little America V, where the Dental Research Laboratory was put into operation.

Saliva was collected monthly for ten months from each member of the thirty-seven-man group, and the same procedures were followed as with their base line studies. An attempt was made to control all factors of collections, such as time of day, time since last eating, and time elapsed since last exposed to the cold outside air.

Data on intestinal concentrations of *Lactobacillus acidophilus* were collected after three months in the Antarctic. This program was initiated as supportive work at this time when the oral picture became interesting. Feces were collected from thirteen members of the original saliva group for five months. These collections, for correlative results, were made at approximately the same time as the saliva collections. Dilutions of 1:1,000 were made, and the intestinal samples were immediately treated in the same manner as the saliva samples.

RESULTS

Oral.—The pre-Antarctic oral *Lactobacillus acidophilus* count taken in November, 1956, at Davisville showed a mean of 16,121 per cubic centimeter per man. Upon arrival in the Antarctic in February, 1957, the group was resurveyed and showed a count of 18,337, which is not considered a significant change. Two months after isolation, the mean group count fell from 18,337 to 5,189. The bacterial count remained depressed at 6,555 until June, when the mean soared to 26,550. The oral *Lactobacillus acidophilus* counts continued to remain high for the remaining five months of the experiment, with means of 20,720, 22,646, 23,103, 17,075, and 15,907 for the months of July through November, respectively. These changes are depicted graphically in Fig. 1.

Using a nonparametric technique[4] for testing the significance of differences, which eliminates the bias due to asymmetrical distributions, the reliability of the difference between monthly bacterial counts was tested. The probability coefficient between February and March was $>.05$, but between February and April and February and May it was $>.01$ but less than $<.02$. The rise noted between May and June was $>.02$ $<.05$, but from June through November, 1957, in all instances between both months and extremes, it was $>.05$.

When the reliability coefficients, which in effect can be considered indices of the amount of measurement error, were tested, they were found in all instances to be greater than .73 when 1.00 was used as the basis of maximum reliability.

Intestinal.—The mean evaluations of intestinal *Lactobacillus acidophilus* counts were as follows: May—6,900; June—24,091; July—7,400; August—24,500; and September—24,490.

Using the same nonparametric statistical technique as was used for the oral study, none of the differences between the monthly fecal *Lactobacillus acidophilus* counts was significant, and there were no significant correlations between salivary and fecal counts, either between the means by month or for individuals.

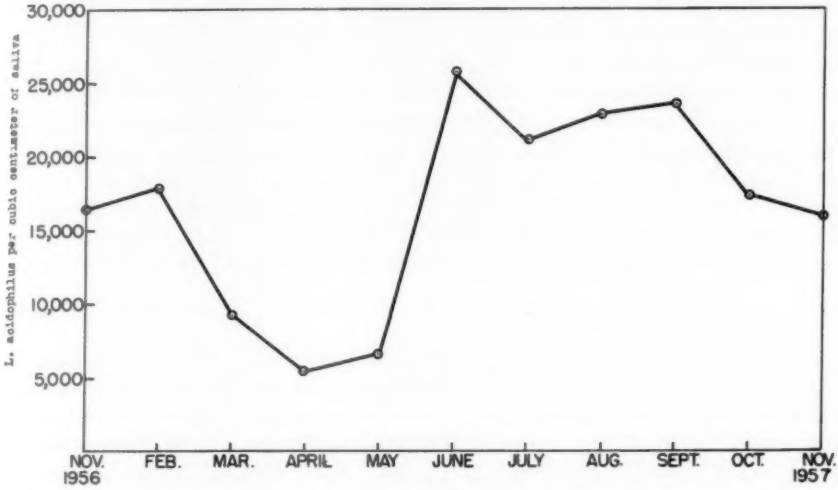


Fig. 1.—Monthly changes in quantities (means) of salivary *Lactobacillus acidophilus* in thirty-seven men living at Little America V in the Antarctic.

DISCUSSION

The fact that bacteria grow optimally at certain temperatures and cease at others is stated in most bacteriology textbooks.[5] Reliable statistics on *Lactobacillus acidophilus* reproduction between these two extremes are not available. However, Tanner[6] reports that Barber, working with *Bacillus coli,* showed that "generation time" gradually increased from 40° C. to 20° C. At 40° C. "generation time" was seventeen minutes whereas at 20° C. it occurred only after sixty minutes. In Fig. 1, there appear to be definite periods in which the population's bacterial counts vary and are statistically different. These differences can be correlated with times of camp activity and specifically with outdoor temperatures.

During the daylight months of February through May, the mean daily temperature ranged from –14.7° F. to –23.4° F., and the relative humidity seldom rose above 5 per cent. The combination of these two factors caused mouth breathing and a decrease of intraoral temperatures. These temperatures were measured on the surfaces of teeth, and an attenuation of bacterial activity in dental plaques has been reported.[7]

It was in these months (February through May) that the majority of the experimental group worked outside. Preparation for the coming winter and continuous daylight resulted in an eighteen-hour work day and a seven-day

week. It was during these months that the *Lactobacillus acidophilus* counts fell to significantly low levels. Long periods of decreased oral temperatures resulted in a reduction in reproductive rates. This, along with the further depletions through swallowing, resulted in lower lactobacillus counts. In June, the worsening weather and continuous darkness forced the men indoors, and they remained there, for the most part, throughout the Antarctic winter. Upon their return to the warm (68° F.) humidified air of their shelters, intraoral temperatures rose and lactobacillus counts increased to and in many instances surpassed pre-Antarctic levels. It was not until the latter part of October that outside work could be attempted on a scale any larger than a "must" basis.

Hemmens[8] has reported that the probable source of intestinal lactobacillus is swallowed saliva. In reporting the work of other investigators, she mentions a correlation between the salivary and stool counts of lactobacilli. Unfortunately, we were not able to support these findings, since there were no significant differences between monthly fecal counts and there was no significant correlation between salivary and fecal counts either by month or for individuals.

CONCLUSIONS AND SUMMARY

Continued exposure to reduced environmental temperatures at low humidities which caused mouth breathing resulted in reduced oral temperatures and lowered *Lactobacillus acidophilus* counts. These counts, retaken a month after the men had been living in a warmer environment, return to original levels. Measurements made on lactobacillus found in stools showed no such correlation with environmental temperature over the period studied. It is suggested that the quantities of lactobacillus counted in saliva do correlate with environmental temperatures.

REFERENCES

1. Medical Report, Deepfreeze I, Addenda B and D, Final Report, Mobile Constr. Batt. (Special), Detachment I, Davisville, R. I., 30 June 1957.
2. Medical Report, Deepfreeze II, Addenda, Final Report, USN Mob. Constr. Batt. (Special), Detachment B, Davisville, R. I., 3 June 1958.
3. Rogosa, M., Mitchell, J., and Wiseman, R.: Selective Medium for the Isolation and Enumeration of Oral Lactobacilli, J. D. Res. **30**: 682, 1951.
4. Wilcoxin, F.: Some Rapid Approximate Statistical Procedures, New York, 1949, American Cyanimid Company.
5. Breed, R. S., Murray, E. G. D., and Hitchens, A. P.: Bergey's Manual of Determinative Bacteriology, ed. 6, Baltimore, 1948, Williams & Wilkins Company.
6. Tanner, F. W.: Bacteriology, ed. 2, Brooklyn, 1933, John Wiley & Sons, chap. 4, p. 72.
7. Stanmeyer, W., and Adams, R.: Reduced Oral Temperatures and Acid Production Rates in Dental Plaques, J. D. Res. **38**: 905, 1959.
8. Hemmens, E. S.: The Relation of Oral and Intestinal Strains of Lactobacilli, J. Am. Dent. A. **37**: 407, 1948.

847 Monroe Ave.
Memphis, Tenn. (R. J. A.)

ANNOUNCEMENTS

American Academy of Oral Pathology

The Academy will hold its fourteenth annual meeting at Northwestern University, Chicago, Illinois, April 27 to 29, 1960. The examination for elevation to Fellowship in the Academy will be given on April 27. Inquiries should be made to Robert J. Gorlin, Secretary, University of Minnesota, School of Dentistry, Minneapolis 14, Minnesota.

Armed Forces Institute of Pathology

The Dental and Oral Pathology Division of the Armed Forces Institute of Pathology in Washington, D. C., announces that its annual course entitled "Pathology of the Oral Regions" will be given March 14 to 18, 1960.

The substance of the course will be directed at the biologic aspects of general dental practice. Attendance is limited to 100, and requests to attend should be made immediately since registration is very heavy. Members of the military establishments should make application in accordance with Circular No. 621-31, Department of the Army, dated June 10, 1959.

Cancer Coordinators

At the 1959 national meeting of the Coordinators of Cancer Teaching at the University of Texas, Medical Branch, in Houston, Texas, on Nov. 12, 1959, the medical and dental chairmen for 1960 were elected.

They are, respectively, Dr. Murray Copeland, Professor of Oncology, Georgetown Medical School, Washington, D. C., and Dr. Robert Gorlin, Professor and Chairman of Oral Pathology, University of Minnesota.

The 1960 meeting will be held in Minneapolis in conjunction with the fourth National Cancer Congress.

Indonesian Dental Association

In commemoration of its tenth anniversary, the Indonesian Dental Association will hold a congress and a trade exhibit in Bandung, Indonesia, during July, 1960. The congress will consist of a business section and a scientific session in which prominent dentists from abroad will be invited to lecture.

For dentists who wish to visit the tourist spots after attending the congress, the beauty of Bandung, Djokja, and Bali will provide an unforgettable vacation. The Indonesian Dental Association will be glad to contact the right tourist agencies for our guests. Information with regard to immigration and currency regulations will be provided on request.

R. M. Soelarko, Congress Secretary
55 Riaustreet
Bandung, Indonesia

University of Alabama

The School of Dentistry will present three refresher courses during the month of February. On Feb. 6 and 7, 1960, Dr. John F. Prichard will instruct a program in "Management of Advanced Periodontal Disease." On Feb. 13, 14, and 15, 1960, Dr. Samuel Gore will present a course, which is limited to practicing orthodontists, entitled "Removable Orthodontic Appliance Construction." On Feb. 27, 28, and 29, 1960, Dr. Daniel J. Holland will offer a course in "Practical Exodontia."

Further information regarding these courses may be obtained from the Director, Refresher Course Program, University of Alabama School of Dentistry, 1919 Seventh Ave. South, Birmingham 3, Alabama.

Boston University School of Medicine

The Department of Stomatology, Division of Graduate Studies, School of Medicine, Boston University and the Massachusetts Memorial Hospitals, announces the following postgraduate courses:

Oral Pathology for Oral Surgeons. Jack Bloom, Kurt Thoma, and Henry M. Goldman. Jan. 11 to 16, 1960. $200.00.

Removable Partial Denture Prosthesis. David J. Baraban. Jan. 21 to 23, 1960. $100.00.

Technics in the General Practice of Dentistry. Henry M. Goldman, David J. Baraban, Leo Talkov, Herbert Schilder, Gerald Kramer, Chester Landy, and Harold Berk. March 7 to 12, 1960. $175.00.

Advanced Course in Periodontology. Henry M. Goldman, Bernard S. Chaikin, Gerald M. Kramer, William Pendergast, and Jack Bloom. March 14 to 19, 1960. $250.00.

Full Denture Prosthesis. Chester Landy. March 28 to April 1, 1960. $150.00.

Periodontal Therapy. Henry M. Goldman, Bernard S. Chaikin, Gerald M. Kramer, William Pendergast, Jack Bloom, and Associates. April 4 to 9, 1960. $175.00.

Apicoectomy—Surgical Endodontic Procedures. Herbert Schilder and Leonard Parris. April 18 to 22, 1960. $175.00.

Periodontal Pathology. Henry M. Goldman and Jack Bloom. April 25 to 30, 1960. $250.00.

Pulp Protection in Operative Dentistry. Harold Berk. May 12 to 14, 1960. $100.00.

Periodontal Prosthesis. Leo Talkov, David J. Baraban, Henry M. Goldman, Bernard S. Chaikin, and Gerald M. Kramer. May 16 to 20, 1960. $200.00.

Surgical Procedures in Periodontal Therapy. Lewis Fox and Associates. May 23 to 27, 1960. $200.00.

For further information, write to Director of Post-Graduate Studies, Department of Stomatology, Massachusetts Memorial Hospitals, 750 Harrison Ave., Boston 18, Massachusetts.

Boston University School of Medicine

The Oral Surgery Section, Department of Stomatology, Division of Graduate Studies of Medicine, announces that applications for the year 1960-61 in oral surgery should be made as soon as possible. The course begins Sept. 15, 1960.

This course consists of lectures and clinical experience in the fundamental medical sciences and a basic review of all phases of oral surgery. Students will also be assigned, in rotation, to the various clinics and the Oral Surgery Out-Patient Department of the Massachusetts Memorial Hospital, and they will assist at oral surgery operations at the Brooks Hospital.

The course has been planned to meet the requirements of the American Board of Oral Surgery, providing a foundation for the clinical internship and residency training leading to qualifications for the practice of oral surgery as a specialty.

The course is under the direction of Dr. Kurt H. Thoma, Professor of Oral Surgery, assisted by a large staff of lecturers and instructors.

Those interested should apply to the Department of Stomatology, School of Medicine, Office of the Dean, Boston University, 80 East Concord St., Boston 18, Massachusetts.

University of Illinois

The College of Dentistry, starting Feb. 1, 1960, will sponsor a three-day seminar on principles of dental teaching.

The seminar, given in cooperation with the Kellogg Foundation, will be presented for dentists who are actively engaged in private practice and who have an interest in methods and principles of dental teaching.

There is no tuition for the course. There will be a registration fee of $10.00.

Additional information can be obtained from Dr. Edward J. Forrest, Associate Dean, College of Dentistry, University of Illinois, 808 S. Wood St., Chicago 12, Illinois.

Northwestern University

The following postgraduate courses are announced by the Northwestern University Dental School:

Current Concepts in Operative Dentistry. Feb. 1 to 3, 1960.
Basic Oral Surgery. Feb. 1 to 5, 1960.
New Concepts in Periodontics. Feb. 1 to 5, 1960. Max Gratzinger.
Pedodontics. Feb. 11 and 12, 1960.
Analysis and Treatment of the Stomatognathic System for the Orthodontist. Feb. 22 to 24, 1960. John R. Thompson.

For further information, address the Director of Postgraduate Study, Northwestern University Dental School, 311 E. Chicago Ave., Chicago 11, Illinois.

Oral Pathology Laboratory of Mount Sinai Hospital

The Oral Pathology Laboratory of Mount Sinai Hospital in New York City will hold its second Clinical Oral Pathology Conference at 5 P.M. on Friday, Jan. 13, 1960. The topic will be "Paget's Disease and Fibrous Dysplasia of the Jaws," and Dr. Lester R. Cahn will be the speaker. The conference will be held in Lecture Room 3-B on the third floor of the Administration Building, Mount Sinai Hospital.

REVIEWS OF THE LITERATURE

REVIEWS OF NEW BOOKS

Transplantation of Tissues. Volume II. Edited by Lyndon A. Peer, M.D., with twelve contributors. Baltimore, 1959, Williams & Wilkins Company. 690 pages. Price, $20.00.

This is the second volume of a most comprehensive and exhaustive review of this fascinating field of surgery. Volume I covered cartilage, bone, fascia, tendon, and muscle. Volume II covers skin, cornea, fat, nerves, teeth, blood vessels, endocrine glands, organs, peritoneum, and cancer cells.

The authors of the various sections are pioneers in this field, and the material is presented in a scholarly and exacting manner. This should not frighten the clinician, however, for the book offers numerous practical applications which can well be adopted by the various surgical specialties.

Now that organ transplanting has been made commonplace by the advances in vascular surgery, we await the break-through which will make these transplants practical. Basic fundamentals, anatomy, current theories, and research are discussed in each of the chapters, with emphasis on specific clinical applications.

This excellent compilation should prove a valuable addition to the literature of this field.

Victor D. Dembrow

Textbook of Oral Surgery. Edited by Gustav O. Kruger, B.S., A.M., D.D.S., F.A.C.D., Professor of Oral Surgery, Georgetown University School of Dentistry, Washington, D. C.; Professor of Oral Surgery, Georgetown University School of Medicine; Chief of Dental Staff, Georgetown University Hospital; Consultant and Member of Executive Committee, District of Columbia General Hospital; Consultant, Veterans Administration Hospital, Martinsburg, W. Va.; Consultant, United States Naval Dental School, Bethesda, Md.; Past President, Middle Atlantic Society of Oral Surgeons; Diplomate, American Board of Oral Surgery. St. Louis, 1959, The C. V. Mosby Company. Price, $12.75.

The editor selected as contributors twenty-one well-known authorities in oral surgery, and each has devoted his efforts to one chapter dealing with a particular phase of the specialty. It is impossible in a short review to do justice to the many outstanding features of the book.

There are 489 illustrations; the reproduction of the roentgenographic material is excellent and the drawings by Mellani are superb. The author is to be congratulated on his choice of illustrations.

This book will be of inestimable value to the student and the practitioner. It is highly recommended by this reviewer.

Occlusal Equilibration and Temporomandibular Joint Dysfunction. Nathan Allen Shore, D.D.S. (Foreword by Clyde H. Schuyler, D.D.S.), Philadelphia, 1959, J. B. Lippincott Company. Price, $12.50.

This is the first full-length book devoted exclusively to the subject of occlusal equilibration and temporomandibular joint dysfunction—usually presented as a subdivision of periodontic practice rather than as a principle basic to all operative dentistry.

It provides the dentist in general practice, as well as the specialist, with complete details on the management of occlusal disharmonies and the treatment of temporomandibular joint dysfunction.

Thomas J. Cook

Implant Dentures: Indications and Procedures. Aaron Gershkoff, B.S., D.D.S., Guest Lecturer on Implant Dentures, Post Graduate Division of Tufts University School of Dental Medicine; Past-President, American Academy of Implant Dentures; and Norman I. Goldberg, D.D.S., Guest Lecturer on Implant Dentures, Post Graduate Division of Tufts University School of Dental Medicine; Past-President, American Academy of Implant Dentures. (Foreword by Irving R. Hardy, D.M.D., Professor of Graduate and Post Graduate Prosthetics, Tufts College Dental School.) Philadelphia, 1957, J. B. Lippincott Company. 256 pages. Price, $12.00.

This is the first text devoted to the subject of implant dentures and it is written by two pioneers in the field.

In this text Gershkoff and Goldberg, who realized the inadequacies of conventional denture construction in certain patients, offer their experiences and techniques.

There are 327 illustrations which are well done and should be of great assistance to those who wish to undertake the implant denture technique.

This book should be of special interest to the prosthodontist and the oral surgeon.

Thomas J. Cook

Dental Roentgenology LeRoy M. Ennis, D.D.S., Chairman of the Department and Professor of Roentgenology, The Thomas W. Evans Museum and Institute, School of Dentistry, and Harrison M. Berry, Jr., D.D.S., Professor of Roentgenology, The Thomas W. Evans Museum and Institute, School of Dentistry. Philadelphia, 1959, Lea & Febiger. Price, $15.00.

This is the fifth edition of a text that first appeared in 1931. As stated in the publication announcement, Berry "brings a new approach and new experience to this already highly authoritative text."

The principal aim of this book has been to organize and set down as concisely as possible dental roentgenographic techniques and the interpretation of films.

This all-inclusive text has 607 pages and 1282 illustrations.

A Textbook of Orthodontia. Robert H. W. Strang, M.D., D.D.S., Director of Courses in Orthodontia, Postgraduate Division, Temple University School of Dentistry, and Director of the Fones School of Dental Hygiene, University of Bridgeport, Connecticut, and Will M. Thompson, Jr., D.D.S., Instructor in Advanced Orthodontia, Postgraduate Division, Temple University School of Dentistry. Fourth edition. Philadelphia, 1958, Lea & Febiger. 880 pages, 1178 illustrations. Price, $20.00.

This book is limited to instruction in the use of the edgewise arch mechanism in the treatment of malocclusion of the teeth. The authors present complete and detailed instruction in clinical orthodontics, as well as a sound theoretical background in this growing specialty.

For this edition Dr. Strang has been joined by Dr. Will M. Thompson. Together they have made a thorough revision of many portions of the text, added a chapter on

"Clinical Cephalometrics" and brought the entire work into line with current advances in this specialty. Every technical description has been tested for accuracy of instruction in teaching, as well as for practical application. The order of procedure in incorporating the various modifications for production of tooth movements is stated specifically. Illustrations of arch wire modifications are improved, and new ones have been added.

Teachers, orthodontists, students, and dental practitioners alike will find this book excellently organized and truly stimulating through every discussion.

Treatment Planning in the Practice of Dentistry. Henry M. Goldman, Lester W. Burket, and associates. St. Louis, 1959, The C. V. Mosby Company. Price, $6.50.

This pocket-sized text of 305 pages, with 95 illustrations, is designated as "The Postgraduate Dental Lecture Series" given at the University of Pennsylvania.

This reviewer happened to be going to the A. D. A. meeting in New York by plane and just could not resist putting this nicely bound book into his coat pocket. It served as a fine traveling companion and is recommended to the student, the general practitioner of dentistry, and the physician.

It could well mark the beginning of a new trend in publications—"pocket size."

Disorders of the Temporomandibular Joint: Diagnosis, Management, Relation to Occlusion of Teeth. Laszlo Schwartz, D.D.S., Clinical Professor of Dentistry, Columbia University, Director, Temporomandibular Joint Clinic, Columbia Presbyterian Medical Center, and eighteen contributors. Philadelphia, 1959, W. B. Saunders Company. Price, $15.00.

This monograph is an all-important contribution to our understanding of temporomandibular joint disorders. The book is divided into five sections: (1) general considerations, (2) diagnostic procedures, (3) differential diagnosis, (4) methods of treatment, and (5) management of disorders.

There is emphasis on the clinical aspects, and case reports are presented in an outstanding manner.

The book has 471 pages and 458 illustrations. It is highly recommended to the dentist and the physician, the general practitioner and the specialist.

Essentials of Removable Partial Denture Prosthesis. Oliver C. Applegate, D.D.S., D.D.Sc., F.A.C.D., Professor of Dentistry (Partial Denture Prosthesis), School of Dentistry and W. K. Kellogg Foundation Institute, Graduate and Postgraduate Dentistry, University of Michigan. Second edition, Philadelphia, 1959, W. B. Saunders Company. Price, $14.00.

The book is divided in two parts. Part I consists of twenty well-presented chapters devoted to a complete coverage of the fundamentals, from the types and uses of various impression materials, through the preparation of the patient for partial denture prothesis, and finally the insertion and balancing of the completed case. All are thoughtfully discussed as a basis for achievement in partial denture service. References in the first part are numerous and authoritative. Instructions for the patient in regard to his responsibility in partial denture maintenance are thoroughly outlined.

Part II is a revision of this section of the first edition, which from clinical experience has been found to be advantageous. This section is devoted to methods now being used by the author.

The entire book is profusely illustrated with 453 illustrations, ten of which are in color. The printing is on semiglossy paper and easily read.

The second edition is not only a desirable text for the undergraduate student but an excellent reference book for the general practitioner who desires knowledge of modern analysis of impression materials, treatment planning, and design.

Howard H. Burkart

ABSTRACTS OF CURRENT LITERATURE

Preanesthetic Medication for Children: Comparison of Oral and Intramuscular Routes. Benjamin Root: Anesthesiology **20:** 49, January-February, 1959.

In this study of 481 children under 10 years of age, a comparison is made of the effectiveness of intramuscular and oral administration of preanesthetic drugs.

Intramuscular scopolamine was effective as a drying agent in a dose of 0.006 mg. per pound, while scopolamine and atropine were usually effective orally in doses of 0.0025 mg. per pound.

Oral meperidine, 1.0 mg. per pound, produced less respiratory depression than intramuscular meperidine in a dose of 0.66 mg. per pound.

Oral premedication was easy to administer, and the children appreciated the fact that they were not going to get a "shot."

T. J. C.

Radiodontic Study of the Mental Foramen. A. Porter Sweet: D. Radiog. & Photog. **32:** 28, 1959.

"Below the second bicuspid on either side, midway between the upper and lower borders of the body of the mandible, is the mental foramen, for the passage of mental vessels and nerve."

This study was made of 600 patients, 164 females and 436 males.

A large number of anomalies were noted as well as different locations where the mental foramen was found.

T. J. C.

Syndrome of Acute Osteomyelitis of the Superior Maxilla in Early Infancy. H. Lieberman and J. Brem: New England J. Med. **260:** 318, February, 1959.

Osteomyelitis of the superior maxilla in early infancy is described, and two cases are presented.

A newborn infant, usually 2 or 3 weeks of age, becomes prostrated and feverish, and swelling of one side of the face more than the other develops, with marked swelling and redness of the orbital region—so much so that the lids can hardly be opened. There is chemosis of the conjunctiva, and when the orbital contents are involved, fixation and proptosis of the globe occur. A purulent nasal discharge, most marked on the involved side, is also present. As the condition progresses, pus may appear in the nose and fistulas may form at the alveolar process of the maxilla, at the canine fossa, or near the inner canthus of the eye on the involved side. Tooth buds may be extruded, with drainage from the alveolar process. In addition to the fever and prostration, vomiting and diarrhea usually appear. In practically all cases in which cultures have been taken, a pathogenic *Staphylococcus pyogenes* var. *aureus* has been recovered.

To minimize or prevent permanent damage, early clinical and bacteriologic diagnosis, surgical intervention when indicated, and treatment with the proper antibiotic, as well as supportive therapy, are essential. Because the infection in this syndrome has a tendency to smolder, it is essential to continue with the antibiotics of choice until it is entirely eradicated.

T. J. C.

Cutaneous and Mucous Membrane Moniliasis. E. R. Seale and J. B. Richardson: Postgrad. Med. **25**: 747, June, 1959.

Moniliasis presents a characteristic clinical picture; it is frequently misdiagnosed and, therefore, improperly treated.

Monilia is the name formerly applied to a genus of fungi now called candida. With few exceptions, *C. albicans* is considered its only pathogenic member. The monilial infection of the tongue and buccal mucosa is called thrush. The diagnosis is made in the laboratory by direct smear and culture.

In the treatment, general supportive and corrective measures are instituted in cases where moniliasis is associated with malnutrition, avitaminosis, etc.

In the treatment of oral moniliasis, gentian violet, 1:5,000 aqueous solution, is used over a period of several days.

The oral suspension of the antibiotic nystatin (Mycostatin) is effective in thrush.

T. J. C.